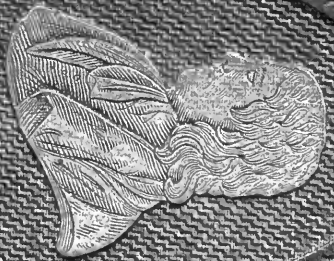

THE NEW SYDENHAM SOCIETY.

INSTITUTED MDCCCLVIII.

VOLUME CVI.

HANDBOOK

OF

GEOGRAPHICAL AND HISTORICAL PATHOLOGY.

BY

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Volume I.—Acute Infective Diseases.

TRANSLATED FROM THE SECOND GERMAN EDITION

BY

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AUTHOR'S PREFACE.

In laying down, now twenty-five years ago, a plan to be followed in the preparation of a Handbook of Geographical and Historical Pathology, I was fully aware of the difficulties that attended both the execution of the work and the introduction of it to the professional public; and, in the preface which I issued with the first part of the book, I spoke of these difficulties plainly and without reserve. The task that I had imposed on myself was not merely to collect and reduce to order an almost unmanageable heap of materials—for the most part a *rudis indigestaque moles*—and to test critically their authenticity and fitness; but it more particularly involved the founding according to a design, and the building up according to a system, of a discipline which had been the subject of but little labour before, and had still to make good its right to a place among the Medical Sciences.

The misgivings which these difficulties gave rise to in me as I approached the execution of my task proved to be only too well justified, in one direction at least, as the work proceeded. Notwithstanding the quantity of material that I had collected, there were considerable gaps which it did not cover; and, more-

over, when I came to test its suitability for the object in view, it contracted into smaller and smaller bulk, so that the book, when it was finished, remained much behind even the modest expectations that I had set out with. I have been more fortunate in overcoming the difficulty that faced me in the other direction—the difficulty of gaining the ear of the learned world of medicine to the subject of which I treated. Their interest declared itself, not merely in the favorable opinion that was soon expressed of my work on almost every hand, but also in the increased attention that has been given since then to the study of Geographical and Historical Pathology. I may therefore permit myself to think that this work of mine, however defective it has always seemed to myself, has provisionally satisfied a want that was felt, and has given an impulse to the further cultivation of this field of medical knowledge; and, in writing now, I may say without boasting that the progress made within the last thirty years by this discipline, with its decided influence on the course of etiological inquiry and its infusion into it of a more scientific character, has been connected in no small degree with the publication of my book.

The start which the geographical and historical study of disease has taken in recent times has left the book more and more out of date; and the desire to go over the ground again, which I began to entertain shortly after the work appeared, has gradually come to be a duty. The distinguished services in the field of Medical Geography rendered by French, English, and American physicians during the last thirty years through the publication of monographs, official reports, or journals specially devoted to the subject—I may

mention in particular the recent series of Health Reports of the War Offices of England and the United States, and the 'Archives de médecine navale'—have brought us much valuable information as to the Climate and Medical Anthropology of many large regions of the globe, which were formerly unknown in these respects, or known very imperfectly. We have also the copious epidemiographical literature of recent years, which has left hardly a single subject relating to Epidemics untouched, and whose treatment of them has been in most cases extremely thorough; this division also, like that of Medical Geography, has its special organs, among which I shall mention the Reports issued by the English Health Department, the Official Sanitary Reports for Sweden, and the 'Transactions of the Epidemiological Society of London.' All such labours have not only added on the grand scale to our knowledge of the distribution of diseases in time and in place, and of the causal connexion between them and the human environment; but, moreover, these researches have been invested with a scientific value such as pertained to only a small part of the earlier writings in the field of Medical Geography; for the standpoint of observers at the present day is completely changed from what it used to be, representing as it does the latest form of pathological opinion. Nowadays we can estimate the health-conditions of many of the most remote parts of the world, in regard to morbid anatomy and etiology, with as much exactness as we were privileged to do not so very long ago, for none but the most civilised States of Europe and North America. It is unnecessary for me to say how much we are likely to profit by this even in our knowledge of those matters of professional practice that concern us nearest home.

Thus it is that Medical Geography and the History of Diseases bears to-day a character quite different from that of the science twenty or thirty years ago. It has filled out in proportions, and acquired finish to an extraordinary degree; and it seemed to me that some attempt to treat of Geographical and Historical Pathology anew was not merely justified, but even called for.

It will be apparent that my task has not consisted merely in amending and amplifying my work of twenty years ago, or in bringing out an edition of it "improved and enlarged." It has involved rather an entirely new treatment of the subject; and, although I have ventured to retain the general ground-plan of the original work, I have allowed a good deal of enlargement on the side of Comparative Pathology, and still more on the side of Etiology; while, on the other hand, I have introduced some restriction in the choice of subjects, or some compression in the treatment of them. Some diseases have so little of geographical and historical interest that they may be appropriately left unnoticed altogether, or disposed of in a passing reference. There are other diseases which have hitherto attracted so little of the attention of observers at various parts of the world that the writings upon them are of the most meagre and defective kind; it was impossible to arrive at a tolerably sound opinion on their historical and geographical position, or on the causal relation between them and the active factors in the etiology, and it seemed advisable, therefore, merely to mention them in order to indicate the gaps in our knowledge, and perhaps to call forth more thorough inquiries and more comprehensive publication.

With these curtailments I shall not find it necessary to go materially beyond the size of the first edition of this Handbook, notwithstanding the expanded treatment of the more important groups of diseases—the Acute and Chronic Infections, the Constitutional Anomalies of general prevalence, the Endemic Morbid Processes (which are so full of scientific interest in many ways), and such Local Affections as have the character of public or national maladies.

I now hand over my work to the learned medical world with the wish that it may meet with as favorable a reception as the first edition of the Handbook has enjoyed. Although I am far from ignoring the many lacunæ and faults that are still present in the work, I can assure the reader that I have laboured according to my strength to make it as complete as possible. I am not without hope that I have made it more available for those circles of practitioners who are accustomed to judge of the worth of a scientific piece of work according to its immediate usefulness—from the so-called "practical" point of view—and who have hitherto regarded Geographical and Historical Pathology as a matter of abstract scientific study lying remote from their needs; available also for those persons whose lofty confidence in their own experience leads them to look upon historical inquiry in Pathology as so much unprofitable dead-weight or as a superfluous luxury, and who thereby succeed in bringing the field of their mental vision within the narrowest possible limits.

I have, in conclusion, a pleasing duty to perform in offering my most obliged thanks to the Directors of Health Departments, of Medical Departments of the Military Services, and of Statistical Departments, as

well as to the large number of Home and Foreign Medical Societies, for their liberality in keeping me supplied with their published reports, and thereby placing me in possession of materials invaluable for the composition of this treatise.

AUGUST HIRSCH.

BERLIN: *June*, 1881.

NOTE BY THE TRANSLATOR.

The English-speaking race are most directly interested in the subject of this book, and their observations in various parts of the world have supplied a large part of the materials for it. Our indebtedness to Professor Hirsch for his self-imposed labours is all the greater on that account. Having now translated the first volume, and transcribed several thousands of references, I can form some idea of the author's task. In a letter, which I venture to quote, he says of the new edition: "Es steckt in dem Buche ein grosses Stück meines Lebens und eine unsagbar mühselige Arbeit, mehr Arbeit als mir aus der ersten Ausgabe erwachsen ist, da das Material innerhalb der letzten zwanzig Jahre sich verzehnfacht hat." The new edition will be in three volumes, of which two have been published. The volume that is now translated came out in the summer of 1881; the second, containing the Chronic Infective, Parasitic, and Constitutional Diseases, in the summer of this year; and I learn from the author that the concluding volume, treating of Local Diseases (or Diseases of Organs), may be looked for in the spring of 1884.

The Index is an addition of my own. Names of places occur so often in the text that it would have made the Index somewhat unmanageable to have inserted every mention of them; I have accordingly given a preference to those where an authority is quoted, and I venture to hope that the Geographical part of the Index will thus serve as a clue to the Bibliography, more especially for those chapters which have no alphabetical list of authors

appended. Professor Hirsch has taken the trouble to read the proof sheets, so as to ensure that the sense of the original shall not have been seriously missed in any passage.

LONDON; *November*, 1883.

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GEOGRAPHICAL

AND

HISTORICAL PATHOLOGY.

INTRODUCTION.

The life of the organic world is the expression of a process called forth and sustained, in organisms that are capable of life, by the sum of all the influences which act upon them from without. The form and fashion of this process, accordingly, are determined by the kind of individuality and by the character of the environment. Each of those two factors shows many differences in time and in space. As regards the human species, the differences are expressed, for the first factor, in the distinctive qualities of generations separated by years, and of races and nationalities scattered over the globe; for the second factor, they are expressed in peculiarities of the climate and the soil, and of the animal and vegetable kingdoms in so far as these are brought into direct relation with man, and further, in the vicissitudes of politics, of social affairs, of the food-supply, and of mental training.

In these considerations lie the germs of a science, which, in an ideally complete form, would furnish a *medical history of mankind,* but which, treated more narrowly and so as to embrace only the pathological side of human life, will give: firstly, a picture of the occurrence, the distribution, and the types of the diseases of mankind, in distinct epochs of time, and at various points of the earth's surface; and, secondly, will render an account of the relations of those

diseases to the external conditions surrounding the individual and determining his manner of life. And this science I have named, from the dominating point of view, the science of *geographical and historical pathology*.

The first attempts to attain, by way of anthropological observations at various parts of the globe, some knowledge of how the particular fashion of human life is dependent upon the peculiarities of the individual and of his surroundings, reach back to the time when medicine was impregnated with the spirit of Hippocrates, whose treatise, '*περὶ ἀέρων, ὑδάτων, τόπων*,' laid down the lines of scientific investigation on this subject. His successors little understood the need of such labours; only in a few of the best Greek and Roman medical authors, such as Celsus, Asclepiades, and Aretæus, do we find here and there indications that they gave some attention to the various effects of climate and diet upon the human organism in health and disease. Such questions were unfamiliar to the physicians of the middle ages; and it was only in the sixteenth century, when distant countries and new worlds were opened up and explored, when the impulse to observe was awakened, and the study of nature came to displace dogmatic speculation, that naturalists and physicians again endeavoured to find matter for scientific investigation in the changing aspects of organic life at various parts of the globe. Along with the animal and vegetable kingdoms, man was included in the scope of these investigations. At first it was things remote and unaccustomed that attracted the attention of observers, but they soon turned to objects near at hand. Thus, botany and zoology, which had made, since Aristotle's treatment of them, no progress worthy to be mentioned, acquired a scientific character; and at the same time research came back, not always consciously, to those inquiries of Hippocrates on the influence of climate, soil, and manner of life upon the habit of the human body. Not only physicians and naturalists, but also many educated travellers, contributed to this record of observations in medical geography and topography,—a record which had reached such proportions towards the end of the eighteenth century, that a systematic recapitulation of it became necessary. The first attempts at a scientific handling of the subject were

Finke's 'Versuch einer allgemeinen medicinischen Geographie,'[1] and Schnurrer's 'Geographische Nosologie.' Not to mention several more special or less important treatises on the subject, these are succeeded by such works of modern date as Mühry's 'Die geographischen Verhältnisse der Krankheiten,'[2] Boudin's 'Traité de géographie et statistique médicales,'[3] and the great work lately published by Lombard, 'Traité de climatologie médicale.'[4]

As the titles of those works indicate, the several authors have taken up different standpoints in their treatment of the subject, the standpoint either of the geography or of the pathology; they have given either a geographically arranged account of the types and of the relations to climate, soil, culture, race, &c., of the normal and pathological processes of life at various parts of the globe, that is to say, a *medical geography* properly so-called; or they have on principle confined themselves to the pathological standpoint, and have directed their investigations to the mode of distribution of diseases over the earth, and to their dependence upon changing external influences—for the most part with a good deal of one-sidedness and with preponderating if not exclusive regard to climate—that is to say, they have taken up the ground of *geographical pathology*.

The same period which produced the earliest of those medico-geographical works, saw also the first attempt at a *historical* handling of pathology, an endeavour to exhibit the comportment and types of diseases within the several epochs of time through which the human race has passed. Investigations in this field received a decided impulse from the outbreak of widespread and severe pestilences, about which men sought in vain for enlightenment in the received authorities of past times—in Hippocrates, Galen, and Avicenna. In this way there was awakened an interest in the study of epidemiology, which the numerous epidemiological works of the sixteenth and seventeenth centuries bear

[1] Leonhard L. Finke, 'Versuch einer allgemeinen medicinisch-praktischen Geographie,' 3 vols., 8vo, Leipzig, 1792—5.

[2] Two vols., 8vo, Leipzig, 1856.

[3] Two vols., 8vo, Paris, 1857.

[4] Two vols., 8vo, Paris, 1877.

witness to; and naturally associated therewith were inquiries designed to discover in the writings of antiquity or of the middle ages, indications of the occurrence of this or that form of pestilence, and to find out how much the physicians of those times knew of the diseases in question. This inquiry was for the most part conducted from a very simple point of view; it was either a kind of search for antiquities or curios, or it took an especially philological direction. It was not given up until towards the end of the eighteenth century, when the enormous quantity of epidemiological material collected from previous centuries gave occasion to a sifting and scientific elaboration, and urged forward the pathological and etiological, rather than the historical, side of the question, and thus gave a different turn to research.

The historical investigation, like the geographical, had from the outset a dual character, inasmuch as it adopted either the chronological standpoint or the pathological; it turned, moreover, towards that side of pathology exclusively, which afforded or seemed to afford especial interest for historical treatment, towards the group of diseases that occurred as epidemics or endemics—the so-called people's plagues (*Volksseuche*). Among the best known writings belonging to the first, or chronological class, are Noah Webster's 'Brief History of Epidemic and Pestilential Diseases,'[1] and Schnurrer's 'Chronik der Seuchen,'[2] works embracing the history of pestilences over the whole globe; and Villalba's 'Epidemiologia Española,' and Ilmoni's 'Bidrag till Nordens Sjukdoms Historia,' which treat of particular regions. The first to take up the pathological standpoint in the history of pestilences was Hensler, the author of the admirable works 'Vom abendländischen Aussatze,'[3] and 'Geschichte der Lustseuche.'[4] Thereafter followed numerous writings on the history of syphilis, smallpox, malignant sorethroat, typhus fever, scarlet fever, and miliary fever; later came Ozanam's 'Histoire médicale génér. et particul. des maladies épidé-

[1] 2 vols., 8vo, Hartford, 1799; Engl. Ed., London, 1800.
[2] 2 vols., 8vo, Tübingen, 1823.
[3] 12mo, Hamburg, 1790,
[4] Vol. i, 12mo, Altona, 1783.

miques,'[1] a work embracing the history of all pestilences; then the numerous classical monographs of Hecker, 'Ueber die Justinianeische Pest,' 'Der schwarze Tod in 14 Jahrhundert,'[2] 'Die Tanzwuth,'[3] 'Der englische Schweiss,'[4] 'Die epidemischen Krankheiten der Jahre 1770-1772' (in his history of modern medicine). Lastly we have the 'Geschichte der epidemischen Krankheiten,'[5] of Haeser, an excellent work in every respect, in which the author, with a correct appreciation of scientific and practical needs, has endeavoured to do justice to the chronological and pathological standpoints equally.

While one fully recognises the diligence in compilation which runs through many of the above-named works, and the great merits of their authors in arranging and critically sifting the mass of material, and while one takes due account of the results to which these investigations have led, yet it is impossible not to feel that they are but the partial labours of pioneers, which we may utilise, in a measure, in our endeavours to reach the goal of all historico-pathological and geographico-pathological inquiry. The full aim and object of such inquiry is to exhibit the particular circumstances under which diseases have occurred within the several periods of time and at various parts of the globe; to show whether they have been subject to any differences, and of what kind, according to the time and the place; what causal relations exist between the factors of disease acting at particular times and in particular places, on the one hand, and the character of the diseases that have actually occurred on the other; and finally to show how those diseases are related to one another in their prevalence through time and through space—a task, the high importance of which for the doctrine of special diseases, for etiology and for hygiene, cannot well be misunderstood or called in question.

[1] 2nd ed., 4 vols., 8vo, Paris, 1835.

[2] Berlin, 8vo, 1832, re-edited by Hirsch, Berlin, 1865. Engl. transl., by Dr. Babington, 12mo., Lond. 1833.

[3] 8vo, Berlin, 1832.

[4] 8vo, Berlin, 1834.

[5] Vol. ii of the 1st and 2nd ed., and vol. iii of the 3rd ed. (1880—82), of his 'Lehrbuch der Geschichte der Medicin und der epidemischen Krankheiten.'

The execution of this task demands obviously, that there shall be a blending of all the points of view previously taken up in historico-pathological and geographico-pathological inquiry ; it demands an extension of the view over the whole field of pathology, and a method of handling whereby the subject investigated may be brought into direct relation with the doctrines of disease. In the present work, which may haply justify the title of a "Historico-Geographical Pathology" that I have chosen for it, I have striven after the attainment of this end in so far as the literary materials at my disposal permitted, and in so far as the scientific or practical interest of the several forms of disease seemed to require.

In arranging the subjects treated of in this work I have followed the order at present in general use in the classification of disease. I accordingly distinguish :

(1) Acute infective diseases.
(2) Chronic infective and constitutional diseases.
(3) Diseases of organs.

I adopt this division without wishing in any way to prejudge or do violence to the facts ; and I do not forget that in an inquiry like the present, it is of less consequence to carry out a strict system (which is indeed not altogether practicable owing to the very partial kind of insight that we have into many of the processes of disease), than to assign definite limits to each of the subjects of investigation.

CHAPTER I.

INFLUENZA.

§1. Influenza takes a prominent place among the acute infective diseases by reason of its wide prevalence in space and in time; the history of the disease may be followed into the remotest periods from which we have any epidemiological record at all, and its geographical distribution, in so far as we may trust the information before us, extends over the whole habitable globe. The leading position of influenza among the epidemic diseases makes it desirable that we should have a survey of the influenza epidemics hitherto known to have occurred; and I have, in the following tabular statement, drawn up a survey of that kind embracing the period from 1173 to 1875. Beyond the year 1173 the epidemiological data, although they certainly relate to influenza, bear a stamp too little characteristic to make them likely to be useful for the following inquiry; and in later periods as well, all those epidemics are left unnoticed which have been erroneously designated by the chroniclers as influenza, and which we must set down as simple bronchial catarrhs widely prevalent at the same season.

Chronological survey of epidemics of influenza according to records existing from the years 1173—1875.[1]

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1173	December .	Italy (1). Germany (2). England (3).
1323	August . .	Italy and France (1).
1328	March . . .	Italy (1).
1387	January . .	Italy (1).
		France: Montpellier (2).
„	March . .	Germany (3).
1404	. . . . .	Germany: Saxony and Thuringia (1). The Netherlands: Flanders (2).
1411	. . . . .	France: Paris (1).

[1] The references to the literature are collected at the end of the section; the numbers placed in brackets beside the names of localities refer to the authorities given there.

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1414	January . .	Italy: Bologna (1), Forli (2), Venice (3), Florence (4).
,,	February. .	France: Paris (5).
1427	September .	France: Paris (1).
1510	. . . . .	General diffusion in Europe (1, 2).
,,	July . . .	Italy: Milan (3).
,,	August . .	France (4). England (5).
1557	. . . . .	General diffusion in Europe.
,,	July . . .	Sicily (1).
,,	August . .	Italy (2): Padua (3), Lombardy (4). Dalmatia (2). Switzerland (5).
,,	September .	France (6): Poitiers (7), Montpellier (8), Nismes (9).
,,	October . .	Netherlands: Harderwyk (10), Alkmaar (11). Spain (12).
1562	Autumn . .	General diffusion in Italy (1, 2, 3).
1580	. . . . .	General diffusion over the East, in Africa and in Europe (1, 2, 3, 4, 5).
,,	June . . .	Sicily (6, 7). Netherlands: Delft (8).
,,	July . . .	Italy (6, 9). France (10, 11). Spain (12).
,,	August . .	Italy (6, 7, 13). Portugal (13). Spain (13a). Constantinople (7). Germany: Augsburg (14).
,,	September .	Germany: Rhine Districts (15), Helmstädt (16), Silesia (17). Hungary (7).
,,	October . .	Germany: Saxony (14, 18, 19), Baltic Coasts (7, 20). Netherlands: Alkmaar (8).
,,	Nov. & Dec.	Denmark, Sweden, Livonia (7).
1591	. . . . .	Germany (1, 2).
1593	. . . . .	General diffusion (1).
,,	June . . .	France (2).
,,	July . . .	Italy: Venice, Rome, &c. (1).
1597	September .	Italy (1).
1626	Winter . .	Italy (1). Germany: Strasburg (2).
1627	. . . . .	Spain (1, 2).
,,	Summer . .	North America: Massachusetts, Connecticut (3). This epidemic, the first that is known to have occurred in the Western Hemisphere, is believed to have spread (according to (4)) to the West Indies and South America (as far as Chili).
1655	June . . .	North America: New England States (1).
1658	January . .	Italy and France (1). Germany: Shores of the Baltic (2).
,,	April . . .	England: London (3).
1675	September .	Germany in wide diffusion: Leipzig (1). France (2).
,,	October . .	Austria (3). Hungary (4). England: London (5).
1688	May . . .	England: London (1).
,,	July . . .	Ireland: Dublin (1).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1693	Oct. & Nov.	General diffusion in Great Britain, the North of France, and the Netherlands (1).
1709	. . . . .	General diffusion in Italy, France, Belgium, Germany, and Denmark.
,,	Jan. & Feb.	Italy: Rome, Florence, Milan, Turin, &c. (1, 2).
,,	April . .	Germany: Berlin (3). Belgium (1).
,,	Summer .	Denmark: Copenhagen (4).
1712	June . .	Denmark: Copenhagen (1).
,,	July . .	Germany: Holstein (2), Jena (3), &c.
,,	August .	Germany: Augsburg (4).
,,	September	Germany: Tübingen (5).
,,	December	Italy: Turin (6).
1729—30	April . .	Russia: Moscow (1).
,,	September	Sweden (1).
,,	October .	Germany: Vienna (2), Upper Silesia (1).
,,	November	Germany: Eisenach (3), Nürnberg, Regensburg (5), Breslau (4). England: London (6), Plymouth (7).
,,	December	England: York (8). Switzerland: Lucerne, Zürich (1).
,,	January	Switzerland: Lusanne (9). Italy: Lombardy, Bologna, Romagna, Toscana (10, 11). France: Paris (1).
,,	February.	Germany: Halle (12), Rhine Districts (11). Switzerland: Rhaetian Alps (9). Italy: Rome, Florence, Monte Casino, &c. (11).
,,	March . .	Italy: Naples, Sicily (11). Spain (13). Iceland (14); the first well-authenticated influenza epidemic there.
1732—33	. . . . .	Seemingly a general diffusion over the globe.
,,	October .	America: New England States, Newfoundland, West Indies, Mexico, Peru (15, 18).
,,	November	Russia. Poland. Germany: Eisenach (1).
,,	December	Germany: Coburg (2). Switzerland: Basel (6). Scotland: Edinburgh (10). Isle de Bourbon (17, 18).
,,	January .	England: London (10, 12), York (13). France: Paris (8, 10), Dijon (9). Netherlands: Leyden (4), Harderwyk (3). Italy: Milan (14).
,,	February.	England: Plymouth (11). Italy: Leghorn (10). Spain: Madrid (10).
,,	March . .	Italy: Naples (19).
,,	April . .	Majorca (16).
1735	. . . . .	Iceland (1).
1737—38	November	England: Plymouth (1). North America (2).
,,	December	West Indies: Barbadoes (2).
,,	January .	General diffusion in France (3).
1742	January .	Germany: Coburg (1), Erfurt (2).
,,	February.	Germany: Dresden, Coblentz (1).
1742—43	October .	Switzerland (2). Italy: Brescia (1).
,,	November	Italy: Milan, Venice (1).
,,	December	Italy: Bologna (1).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1742—43	January . .	Italy: Rome, Pisa, Leghorn, Florence, Genoa (1).
„	February. .	Italy: Naples, Sicily (1). France: Paris (6), and in general diffusion.
„	March. . .	Netherlands: Leyden (3). Belgium: Brussels (4).
„	April . . .	England: Plymouth (5).
1757—58	September .	General diffusion in North America (1). West Indies: Barbadoes (2).
„	December .	France: Boulogne (9).
„	May . . .	France: Lille (8), Paris (7).
„	September .	Scotland: Edinburgh (4).
„	October . .	Scotland: Fife, St. Andrews, Perth, Inverness, Aberdeen, &c. (4, 5, 6). England: York (3).
1761—62	Dec.—May .	General diffusion in North America and West Indies.
„	February. .	Germany: Breslau (10), &c.
„	March. . .	Germany: Vienna (3, 4) and Hungary. Denmark: Copenhagen (13).
„	April . . .	Germany: Magdeburg, Hamburg (10), Bremen (4). Great Britain: London (10, 11), Edinburgh (12). Italy (2, 3).
„	May . . .	Ireland: Dublin (13).
„	June . . .	France: Alsace (6, 14), Lille (8).
„	July . . .	France: Nismes (6), English Fleet in the Mediterranean (10).
„	September .	France: Cusset (9).
1767	. . . . .	Widely diffused over North America and Europe.
„	April . . .	North America: New England States (10). Germany: Eisenach (1), Giessen (2).
„	June . . .	England: London (7).
„	July . . .	France: Lille (4).
„	August . .	France: Paris (3).
„	October . .	France: Provence (5), Normandy (6). Italy: Toscana (12).
„	November .	Italy: Naples (8).
„	December .	Spain: Madrid (9). Cayenne (11).
1772	February. .	North America (1).
1775—76	March. . .	Germany: Clausthal (1).
„	June . . .	Germany: Vienna (2).
„	September .	Italy: Naples (10).
„	October . .	Italy: Pisa (10). France: Paris (3), Bourges (5), Bruyères. England: London (7), York (9). Ireland: Dublin (11).
„	November .	France: Bordeaux, Lyons, &c. (3). Wide diffusion in England (8, 9).
„	December .	France: Normandy (4), Montpellier (6). England: Devonshire (9).
„	January . .	France: Martigues, Poitiers, Brest (4).
1780—81	January . .	France: Paris (3) and general diffusion (2).
„	March. . .	France: Lorraine (4). Italy: Milan, Turin, &c. (5). Germany: Heidelberg (1). Brazil (6).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1780—81	December	Russia: St. Petersburg (7).
,,	February	Russia: Wilna (7).
,,	March	North America (8).
1781—82	. . . . .	General diffusion over the Eastern Hemisphere.
,,	Autumn	China, British India (31, 32).
,,	December	Siberia. Russia: Kasan (5, 31).
,,	January	Russia: St. Petersburg (4).
,,	February	Russia: Reval (4, 36), Riga (4, 3). Finland: Lovisa (4). Germany: East Prussia, Tilsit, Braunsberg, &c. (7).
,,	March	Germany: East Prussia (7), West Prussia, Pomerania (1).
,,	April	Denmark (4, 20, 25). Germany: Nördlingen (4), Erfurt (13, 14), &c. (1, 2, 12). Sweden (4). England: Newcastle (28, 31). Hungary: Miskolcz (23).
,,	May	Germany: Prague (20—22), Hamburg (9), Clausthal (10), Lauterbach (11), Mayence (15), Giessen (16), Vienna (22), Nürnberg (17), and other places in Central and Southern Germany. England: London (27, 29, 30), Devonshire (34), and various parts of Suffolk, Surrey, &c. Scotland: Edinburgh and elsewhere (23).
,,	June	Germany (South): Freiburg (19), &c. Netherlands (27). England and Scotland generally (2, 34). France: Alsace, Flanders, Brittany (4, 37, 38), Paris (36), &c. Italy: Sinigaglia, Ancona, Urbino, Spoleto, Rome, Florence, Parma, Modena, Bologna, Venice, Pavia, Verona, Milan (4, 39—42).
,,	July	France: Orleans, La Rochelle, Montpellier, &c.
,,	August	Spain: Madrid, &c. (4, 43).
1788	March	Russia: St. Petersburg, Kherson, Poland (8).
,,	April	Germany: Vienna (3). Hungary: Miskolcz (2).
,,	May	Denmark: Copenhagen (11).
,,	June	Germany: Munich (1). England: Plymouth (7).
,,	July	England: London, Kent, Dover (5), Bath (4), Manchester (6).
,,	August	England and Scotland: Cornwall, Montrose (5). France: Paris (9). Italy: Padua and other places (12).
,,	September	France: Lille (10). Italy (12).
,,	October	Italy: Verona, Brescia, Mantua (13). Switzerland: Geneva (14).
1789—90	. . . . .	General diffusion over the Western Hemisphere (1, 3).
,,	September	North America: Georgia (4), New York (1, 2).
,,	October	North America: Philadelphia (3), New England States (1). West Indies: Jamaica (6).
,,	November	North America: Massachusetts (4). West Indies (3, 5).
	December	Nova Scotia (4). South America (3).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1789—90	Spring . .	North America: Albany, Vermont, Boston, New York, and various other places anew (1).
1798	Nov. & Dec. .	General diffusion in U.S. of America.
1799 } 1800 }	. . . . .	North-eastern parts of Europe.
,,	October . .	Russia: Moscow (2).
,,	November .	Russia: Vologda, Archangel (2).
,,	December .	Russia: Riga, Mittau (2), Kasan, St. Petersburg (1), Ukraine, Volhynia, Podolia (10).
,,	January . .	Galicia: Lemberg (10).
,,	February . .	Poland: Warsaw (11). Germany: Province of Prussia (2).
,,	April . . .	Germany: Vienna (9), Posen (2).
,,	May . . .	Denmark: Copenhagen (12).
1800—1	September .	China (14).
,,	October . .	Germany: Lüneburg (4). France: Lyons (13).
,,	November .	Germany: Altenburg (5), Paderborn (6), Donaueschingen, Sigmaringen.
,,	December .	Germany: Stuttgart (8).
,,	January . .	Germany: Görlitz (7).
1801	. . . . .	Brazil: Rio Janeiro (1)
1802—3	Winter . .	Very extensive diffusion in France (2, 3, 4, 14, 15, 16, 17).
,,	January . .	Germany: Stolberg-on-Rhine (7). Italy: Milan (18, 19). England: London, &c. (10, 11, 13).
,,	February .	Germany: Frankfort-on-Main (5), Cologne (6), Mayence (8), Hanau (23). Widely spread in Britain (12, 13, 22).
,,	March . . .	Universally in Britain (12, 13). Italy: Genoa (20), Verona, Padua, Modena (21). Switzerland: Geneva (24).
,,	April . . .	Germany: Paderborn (9). Britain (13).
1804	. . . . .	Iceland (1).
1805—6	Winter . .	Generally in West Indies (13); in St. Bartholomew in November.
,,	,, . .	Spain: Catalonia (12).
,,	September .	Russia: Wilna (3).
,,	November .	Germany: Erlangen (2). France: Paris (4, 6, 9, 10). Narbonne (7).
,,	January . .	France: Versailles (5), Lyons (15). Italy: Lucca and all Northern Italy (11, 14).
1807	. . . . .	General in North America (1).
,,	February .	Massachusetts (2).
,,	Spring . .	New England States (1, 3, 7).
,,	October . .	Western States (1).
1807—8	November .	Scotland: Edinburgh (3).
,,	December .	England: London (1), Nottingham (2).
,,	January . .	England: Newcastle (4).
1811	. . . . .	General diffusion in Brazil (1).
1815—16	Autumn and Winter }	General diffusion in North America (1).
,,	September .	Boston, U.S.A. (1).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1815—16	October . .	New York (3).
,,	Winter . .	Pennsylvania (2).
,,	February . .	South Carolina (1). Iceland (5). Widely spread in Brazil (4).
1824—25	. . . . .	In the Northern States of the Union (1).
,,	October . .	Boston.
,,	November .	New York.
,,	December .	Philadelphia.
1826	. . . . .	Widely spread over the Western Hemisphere.
,,	January . .	U. S. of America: general in the Southern States, Georgia (1), Alabama (3).
,,	February . .	U. S. of America: general in the Atlantic States as well as in the Eastern, Northern, and Western (1).
,,	May . . .	Generally in Mexico (2).
,,	September .	Extensively in Peru (4).
1827	. . . . .	Generally diffused in Siberia and Eastern Russia (1).
,,	January . .	Siberia: Tobolsk, Tomsk.
,,	February . .	Russia: Perm.
1830—32	. . . . .	General diffusion over the Eastern and Western Hemispheres.
1830	January . .	China (1, 49).
,,	September .	Manilla (1). Polynesia: Navigator's Islands (47). probably for the first time.
,,	November .	Russia: Moscow.
1831	January . .	Russia: St. Petersburg (4). East Indies: Borneo, Sumatra (2).
,,	February . .	Russia: Courland (5), Dorpat (5).
,,	March . . .	Poland: Warsaw. East Indies: Java (2, 3).
,,	April . . .	Germany: East Prussia (11), Silesia (15).
,,	May . . .	Germany: Danzig (10), Brandenburg (12, 13), Berlin (51), Magdeburg (14), Kingdom of Saxony (16, 17), Duchy of Nassau (54), Rhine districts, in parts (18, 19), Homburg, Bamberg (27), Bohemia (29, 31), Vienna (30), Würtemberg, in parts (25). Finland (7). Denmark (8, 9).
,,	June . . .	Germany: Rhine districts, Cologne (20), Hanau (22), Mayence (23), Ansbach (28), Heidelberg (24), Würtemberg, in parts (25). Belgium (53). France: Paris, &c. (34—36). Sweden, in parts (7). Scotland: Glasgow. England: Douglas, Isle of Man (39). Further India: Singapore (3). East Indies (3a).
,,	July . . .	Germany: Aix (21), Würtemberg (25, 26). Switzerland: Geneva (32, 33). France: Toulouse (37). England: London, &c. (38). Sweden, in parts (7). Further India: Penang (3).
,,	November .	Italy: Rome (41, 42), Toscana (48). U. S. of America: New Jersey (44), Philadelphia (50).
,,	December .	Italy: Naples and Sicily (40, 43, 48, 52).
1832	January . .	Spain: Gibraltar. North America: Philadelphia (44), apparently a revival of the previous epidemic.

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1832	February.	North America: Georgia (45).
,,	April . .	India: Indore, Meerut, and other places (46).
,,	December	India: Bangalore (46).
1833	. . . . .	General diffusion over Western Asia, North Africa, and Europe.
,,	January .	Russia: Moscow, Perm, Kasan, St. Petersburg (3), Riga (4).
,,	February.	Russia: Odessa (2). Galicia: Brody (63). Germany: Memel (24).
,,	March . .	Egypt (1, 4). Syria (4). Poland: Warsaw (15). Germany: Prov. Prussia (15—19, 24), Posen (24), Oppeln, Breslau (22, 23), Prov. Brandenburg and Berlin (25—33), Lüneburg (39), Bohemia (60). Denmark: Elsinore (9).
,,	April . . .	Germany: Greifswald (20, 31), Liegnitz (22), Prov. Saxony (34), Hamburg (36), Kingd. of Saxony (40—42), Jena (43), Fulda (44), Homburg (56), Bohemia (61, 62), Lower Austria and Vienna (64—67), Upper Austria and Linz (68, 69). Hungary: Pesth (74, 75). Denmark: Copenhagen (9—13), Fünen and Jütland (9), Alborg (14). France: Paris (79—82), Bordeaux (85), &c. Britain: London (93, 94), Birmingham (95), Edinburgh (96), Armagh (97).
,,	May . . .	Germany: Schleswig-Holstein (35), Bremen (37), Marburg (45), Rhine Province (46—49), Duchy of Nassau (100), Heidelberg (50), Würtemberg (51), Munich (53, 54), Weissenberg (55), Würzburg (57, 58), Kirchenlamitz (59), Tyrol (70), Styria (72), Laibach (71). Dalmatia (73). Italy: Lombardy, Venetia (86—88, 98).
,,	June . . .	Germany: Ditmarschen (35), Osnabrück (38), Würtemberg, &c. (51). Netherlands: Amsterdam (99). Italy: Modena, Romagna, Ancona (90—98).
,,	July . . .	Germany: Sigmaringen (52). Italy: Novara (89).
,,	September .	Switzerland: Aarau (76), Zürich (77), Bern (78). France: Dpt. de la Moselle (84).
,,	November .	Italy: Naples (91, 92—98), Sicily (98).
1834	January . .	Cayenne (1).
,,	February. .	India: Calcutta (3).
,,	December .	Brazil: Rio de Janeiro (2).
1836—37	. . . . .	Considerable diffusion in the Eastern Hemisphere.
,,	October . .	Australia: Sydney (1).
,,	November .	South Africa: Cape Town (2). Java (96). Further India: Penang (104).
,,	December .	Russia: St. Petersburg (3). Sweden (4). Denmark: Elsinore, Copenhagen, &c. (5, 8). Germany: Greifswald (10), Province of

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
		Brandenburg, Berlin (16, 17). England, at various places (46).
1836—37	January . .	Egypt, Syria (91, 92). Denmark: Bornholm, Viborg, &c. (2). Germany: Prov. Prussia (9), Schleswig-Holstein (11), Hamburg (12—15), Breslau (18), Peitz (19), Kingdom of Saxony (20, 21), Fulda (23, 24), Hesse (25, 27, 28), Cassel (99), Rhenish Prussia (30, 32, 33). Würtemberg (34). England (46): London (47, 48), Sidmouth (103), Birmingham (49), North Shields (50), Liverpool (102). Ireland (51) France: Paris (54—65), Strasburg (66), Rennes (68), Nancy (69), Bordeaux (81), &c. Netherlands (97). Switzerland: Geneva (101).
,,	February . .	Germany: Kingdom of Saxony (21), Jena (22), Emden (29), Rhenish Prussia (30, 31), Nassau (98), Würtemberg (34), Stuttgart (35), Kreuzwertheim, Bavaria (37), Lower Austria (38—41). Switzerland: Solothurn, &c. (43). Belgium (95): Antwerp (53). France: Dpt. de la Moselle (70), Lyons (71—75), Dijon (80, 81), Narbonne (76), Toulouse (77), Dpt. Tarn et Garonne (78), Montpellier (82). Northern Italy: Turin (83), Brescia (84), &c. (85—88). Spain and Portugal (100): Lisbon (89, 90).
,,	March . . .	Germany: Würtemberg (34), Sigmaringen (36), Salzburg (42). Switzerland: Uster, Höngg, Bern, &c. (43—45).
,,	July . . .	Faröe Islands (5). Mexico (93).
1838	February .	Isle de Bourbon (1). Iceland (3, 4).
,,	November .	Australia: Sydney. New Zealand (5).
1839	. . . .	Abyssinia: Tigré (2).
1841	January . .	Germany: Prov. Prussia (1), Halle (2), Kingdom of Saxony (3), Lüneburg (7).
,,	February .	Germany: Peitz (4), Fulda (5), Jena (6).
,,	March . . .	Germany: Province of Westphalia (13), Nassau (13). Vienna (10). Hungary: Pesth (9).
,,	April . . .	Germany: Rhenish Prussia (8). Ireland: Dublin (11, 12).
1842	January . .	Belgium (1).
,,	March . . .	England: London (4), York (5), &c.
,,	Spring . .	France: Paris (2, 3). General diffusion in Egypt (6). Chili (7).
1843	March . . .	Germany: Berlin (5), Westphalia (3). England: London (4). Iceland (11).
,,	April . . .	France: Paris (5).
,,	May . . .	North Siberia (1).
,,	Summer . .	General diffusion in North America (9).
,,	June . . .	U. S. of America: New England States, New York, Western States (8).
,,	July . . .	U. S. of America: Pennsylvania (10), Central and Southern States (8). Virginia (12).
,,	August . .	U. S. of America: S. Carolina (8).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1844	January . .	Germany: Rhenish Prussia (1), Westphalia (2), Nassau (7). England: Cheshire (3).
,,	February. .	France: Dpt. Allier (4). Switzerland: Geneva (8).
,,	November .	Russia: St. Petersburg (5). Wide diffusion in Cayenne (6). New Zealand (9).
1845	January . .	Germany: Regensburg, Minden (1). Switzerland: Zürich, Bern (2).
1846—47	Winter . .	England: Richmond (1). Denmark (2). Belgium (4). Switzerland: Basel (5). France: Paris (10).
,,	February. .	Switzerland: Bern (7). France: Toulouse (8). Russia: Jaroslav (6).
,,	March . . .	Russia: St. Petersburg (3).
,,	August . .	Turkey: Constantinople (9).
1847—48	. . . . .	Generally diffused over the Eastern Hemisphere.
,,	September .	France: Rennes (23).
,,	October . .	France: Lyons (24). Denmark: Copenhagen (25). Germany: Bohemia (2).
,,	November .	Germany: Bohemia (2), Schleswig-Holstein (1), Würtemberg (4). Denmark (5). Netherlands (6): Amsterdam (26). France: Marseilles, &c. (9). Britain: London (10, 11, 12), York (13), Edinburgh (15, 16, 27).
,,	December .	Germany: Erlangen (3), Duchy of Nassau (28). Netherlands (6, 7). Switzerland (8). France: Puy de Dôme (17), Paris (29). Northern Italy: Genoa, Nice, Alessandria, &c. (9. 18, 19). Spain: Barcelona, Madrid (9). Greece, Egypt, Algiers (20). North of Scotland (14).
,,	January . .	Germany: District of Holzen, Bavaria (30). Belgium: Liege (31). Southern Italy: Naples (9). Hawaïan Islands (32).
,,	Oct. & Nov. .	West Indies: St. Vincent (1), Santa Cruz, &c. (21).
1850—51	. . . . .	Generally diffused over the Western and Eastern Hemispheres.
,,	December .	West Indies: Martinique (1). America: Peru and Chili, spreading from Lima along the coast to Valparaiso (2).
,,	January . .	North America: California (3). Germany: Osnabrück (4), Duchy of Nassau (5). Prague (13).
,,	February. .	Germany: Upper Harz (6). Sweden (7).
,,	March . . .	Germany: Bavaria (8). France: Paris (9, 10). Italy: Asti (11). Egypt (12).
1852	. . . . .	Australian Continent and Tasmania (1). South America: Peru and Chili, as in previous year (2).
1853	January . .	Cape Colony (1).
,,	May . . .	Faröe Islands (2).
1854	May . . .	Bavaria, in several departments (1).
1855	. . . . .	General prevalence in Europe.
,,	January . .	Russia: St. Petersburg (1). Germany: Bavaria (2), Würtemberg (3). Netherlands (4). Belgium: Liege (5). Italy: Naples (6).

Year.	Season of the Epidemic.	Area of Epidemic Prevalence.
1855	March . . .	Germany: Duchy of Nassau (7).
,,	May . . .	Iceland (8).
,,	June . . .	Brazil: Rio de Janeiro (9).
1856	February . .	Faröe Islands (1).
,,	July . . .	Iceland (2).
1857—58	. . . . .	Wide diffusion over the Western and Eastern Hemispheres.
,,	August . .	Central America: Panama (1).
,,	September .	West Indies (2, 3). South America: Coasts of Chili and Peru (3). British North America: Vancouver's Island (3).
,,	December .	Russia: Courland (4), St. Petersburg (5), Samara (6). Germany: Duchy of Nassau (7), Würtemberg (8), Bavaria (9), Bohemia (17). Belgium (10). France generally (11): Paris (12), Strasburg (13), Lyons (14).
,,	January . .	Italy: Naples (15).
,,	May . . .	Faröe Islands (16).
1860	June & July .	Australian Continent and Tasmania (1, 2).
1861	December .	North America: Philadelphia (1).
1862	January . .	Bermudas (1). Netherlands: Rotterdam (2).
,,	April . . .	Cape of Good Hope (3).
,,	May . . .	Iceland (4).
1863—64	Winter . .	New Caledonia (1). France: Paris and elsewhere (2). California, along the Pacific coast (3).
,,	February . .	Switzerland: Canton Zurich (4).
1866	March . . .	France: Paris, Dpt. de l'Aisne (1), Bains en Vosges (2).
,,	May . . .	England: London (3).
,,	December .	Réunion and Mauritius (4).
1867	February . .	France: Paris (1, 2, 3), Strasburg (4).
,,	March . . .	Germany: Stuttgart (5).
,,	April . . .	Belgium: Ghent (6).
1868	December .	Turkey: Constantinople (1).
1871	December .	Cape of Good Hope (1).
1873	Jan.—March	Universally in North America: Pennsylvania, Ohio, Virginia, Illinois, Iowa, Michigan, Wisconsin, Minnesota, Missouri, Alabama, Louisiana, Texas, and other States (1).
1874—75	Winter . .	Widely spread over the Western and Eastern Hemispheres. North America: Texas, New York, South Carolina, Ohio, Iowa, and other States (1). Germany: Austria (2). France: Paris, Bordeaux, Toulouse, Havre, &c. (3), Dpt. des Ardennes (4), Lyons (5). Sweden (6).

§ 2. Always occurs as an Epidemic or Pandemic.

Influenza always occurs as an *epidemic* disease, whether within a narrow circle or even confined to particular places, or in general diffusion over wide tracts of country, over a whole continent, and, indeed, not rarely over a great part of the globe as a true *pandemic*. It is in this last respect that influenza takes an exceptional place among the acute infective diseases; no other of them has ever shown so pronounced a pandemic character as influenza. In estimating the distribution in space to which the disease has attained in the several epidemics, it should be kept in mind that, for many of them, the records available are but defective ones, not warranting definite conclusions as to the area of epidemic distribution. And this holds good not only for past centuries, but still more for the decades just expired and in particular for the pandemics of the years 1857-58, and 1874-75. The interest in the subject had become, exhausted for the greater number of observers and chroniclers, or, at all events, it has not been aroused except in the case of especially severe and destructive epidemics; and one may therefore premise that, in many cases in which there seem to have been only isolated outbreaks of the disease at various points, there have been intermediate outbreaks not recorded; so that the conclusion as to the extent of the epidemic is not altogether reliable. Beyond doubt, influenza has prevailed in tropical and sub-tropical regions much oftener and much more widely than the somewhat scanty information from those parts would lead us to believe; and it is therefore justifiable to surmise that the disease as a pandemic has even greater importance than we might ascribe to it from the data before us.

In truly pandemic form, we meet with authentic influenzas in the years 1510, 1557, 1580, 1593, 1732-33, 1767, 1781-82, 1802-3, 1830-33, 1836-37, 1847-48, 1850-51, 1855, 1857-58, and 1874-75; in several of these pandemics, the disease extended not only over the Eastern Hemisphere, but it reached also to the Western; in others it remained limited to the former; while pandemics are known to have occurred exclu-

sively in the Western Hemisphere in the years 1647, 1737-38. 1757-58, 1761-62, 1789-90, 1798, 1807, 1815-16, 1824-26, 1843, and 1873. Widely prevalent influenzas over large tracts of country occurred in Germany in 1591, 1658, 1675, 1800, 1841, and 1844; in France in 1737, 1775, 1779; in the West Indies in 1805.

Among isolated outbreaks of the disease, the often observed *epidemics on board ship* are especially interesting. In several cases of the kind, the crews were attacked, and that too just as suddenly and without warning as when influenza appears on land, while the ships were lying in port or cruising off the coast, no trace of the disease having shown itself either before or after in the same region ashore. Among the observations to that effect may be mentioned: that of Pop,[1] on an epidemic in February, 1856, on board a Netherlands frigate in the harbour of Macassar, in which 144 out of a ship's company of 340, fell ill in a few days; the observations by surgeons of the English navy[2] on the breaking out of influenza in August, 1856, in the harbour of Rio de Janeiro, and, in October of the same year, in the harbour of Callao; those by surgeons of the German navy[3] on the appearance of the disease on board a ship of war in April, 1875, in the North Pacific (38° N., 149E.) a few days after leaving Yokohama, and on board another ship in July of the same year in the Gulf of Pe-chili, China; and lastly by Chaumezière,[4] on the epidemic of influenza on board a French frigate in February, 1863, four days after leaving the harbour of Gorée, Senegambia: not a trace of the disease had shown itself in the town; while another ship of war, that left Gorée two days earlier, and took the same course, arrived in the harbour of Brest without having had *a single case* of influenza on board. In other and still more interesting cases, the disease has appeared, at a time when it was generally prevalent on land, among the crews of ships on the high seas which had not previously communicated with an infected shore; and those

[1] 'Nederl. Tijdschr. voor Geneesk,' 1859, iii, 22.

[2] 'Statist. Report on the Health of the Navy for the year 1856,' p. 100.

[3] 'Statist. Sanitätsbericht über die kaiserl. deutsche Marine für das Jahre 1875-1876,' 22.

[4] Chaumezière 'Fièvre catarrhale, épidémie observée à bord du vaisseau le Duguay-Trouin aux mois de Févr. et Mars, 1863,' Paris, 1865.

outbreaks befell at the same time as the outbreaks of influenza on the coasts nearest to the position of the ships. Thus, in September, 1781, influenza attacked the crew of an East Indiaman on the voyage from Malacca to Canton, so generally that scarcely a single person escaped; when they left Malacca, the disease was not prevalent there, but when they arrived at Canton it transpired that their outbreak on board, in the China Sea, had happened at the very time when the disease was showing itself with equal intensity at Canton.[1] On board the fleet of Admiral Kempenfeldt, which had sailed from Spithead on the 2nd of May, 1782, influenza broke out at the end of the month with such intensity that the ships were obliged to return to port in the second week of June; they had not been in communication with any part of the coast, but had been cruising on the high sea between Brest and the Lizard. Similar observations were made, at the time of the universal prevalence of influenza in 1837, on board English ships of war cruising on the coasts of Spain and Portugal, and in Indian waters.[2] Renault[3] records an epidemic of influenza in 1847, on board a French mail steamer on the passage from Marseilles to Alexandria, coincident with the outbreak of the disease on the Mediterranean coasts. On board an English ship of war cruising on the coast of Cuba in 1857, and holding no communication with the shore, the greater part of the crew fell ill of influenza; it was afterwards ascertained that the disease had been prevalent during the very same time at Havanna, in Trinidad, and in other West India islands.[4] For the same year (1847) we have the following from the Pacific station:[5] "Influenza broke out in the *Monarch* while at sea, on the passage from Payta to Valparaiso. She had left the former place on the 23rd of August and arrived at the latter on the last day of September. About the 12th of the month. . . seven

[1] This fact, and the one that follows, are taken from communications by surgeons of the English navy, which are collected in Himly's 'Darstellung der Grippe vom Jahre 1782, &c.,' Hanover, 1833, p. 8.

[2] 'Statist. Report on the Health of the Navy for the years 1837—1843,' ii, 8, iii, 7.

[3] 'Gaz. Med. de Paris,' 1856, 680.

[4] 'Report on Health of Navy for year 1857,' 41.

[5] Ib. 69.

men were placed on the sick list with catarrhal symptoms, and during the following ten days upwards of eighty were added. . . . On the arrival of the ship at Valparaiso, the place was healthy; but, in the course of a few days, some cases of influenza made their appearance, and very soon afterwards the disease extended over the whole town. . . The surgeon further observes that the whole coast, from Vancouver's Island southwards to Valparaiso, was visited by the epidemic."

§ 3. Mode of Progression.

There have been two points which have always attracted the particular attention of those who have investigated influenza with reference to its time of outbreak and its range of diffusion: viz. *periodicity in the recurrence of epidemics or pandemics;* and the order in which particular places, districts, or larger tracts of country have successively been invaded by the pandemic, or in other words, *the progress of the disease in a definite line from east to west.* As regards the first-named point, a glance at the chronology of influenza pandemics is sufficient to convince one that the assumption of a definite periodicity in their succession is absolutely groundless, and that any such regularity in the more isolated cases of its epidemic prevalence in various parts of the globe is in nowise capable of proof.[1] With apparently greater justice it has been inferred from the data of earlier observers, that there is a regular progress of the sickness from east to west. This notion is based upon an assumption, to my mind erroneous, that the separate portions of a great epidemic or pandemic of influenza stand in a direct pathogenetic relation to one another, and that the causative agent, beginning at one point and radiating therefrom, has extended to ever wider and wider circles. It cannot certainly be denied that

[1] Finsen ('Jagttagelser angeaande Sygdomsforhold i Island.' Kjöbenh. 1874, 24) writes in this connexion respecting Iceland: "Those who rely upon the experience of former times assume that this infective disease breaks out every nine years, but that inexplicable periodicity has not been kept up in more recent times."

some pandemics, regarded as a whole, do afford evidence of the sickness travelling in the alleged direction, from east to west; in other cases, however, the disease has gone in an opposite direction,[1] or it has travelled from north to south, or from south to north. Still less is there any definite track of sickness discoverable within the more isolated and smaller circles. Not unfrequently, as for instance in the pandemics of 1833 and 1837, many countries taken together in a group have been smitten by the disease as if *at one blow;* while, for particular localities situated within that area, there have been intervals of one or more weeks in the time of the influenza appearing; and there are not wanting observations to show that the sickness had raged for a considerable time in one quarter of a town before it showed itself in the others. *The larger number of facts is rather in favour of a radial progress of influenza, or a progress by leaps and bounds, than of a linear progress; while, in a comprehensive review of the facts, the direction is found to be sometimes to one point of the compass, sometimes to another.* So that the extension of the disease in one single direction cannot be regarded as a peculiarity appertaining to influenza.

A few facts from the histories of outbreaks may serve to elucidate the questions here raised. As early a writer as Baker[2] says, with respect to the spread of the disease in England: "I have considered it worth while to inquire whether this epidemic catarrh has passed from one part of our island to another in any definite line of progress; whether, in fact, it has travelled from east to west, or from northern localities southwards, or whether it has made the journey in the opposite order. But I have ascertained that it obeyed no law whatsoever, running its uncertain course in a somewhat desultory manner." In the epidemic of 1833, the disease in the department of Königsberg, according to Richter[3] "followed no definite geographical line, but attacked here one place there another, as if it had been leaping from island to

[1] Gluge thinks that he has discovered the spread of influenza previous to the 17th century always to have been in a direction from west to east; but the scanty and in part unreliable data concerning the influenza pandemics of that period hardly warrant the formulating of such general laws.

[2] 'Opuscula medica,' Lond., 1771, 29.

[3] L. c., 120.

island." In the epidemic of 1837, the districts of Denmark lying to the west suffered, according to Bremer (l. c.), quite a month before those to the east. On the continent of North America, the extension of the disease is just as often from south to north (epidemics of 1761, 1789, 1826) as from north to south (epidemics of 1790 and 1815), and not unfrequently (epidemic of 1843) it proceeds in an altogether irregular manner. In Greenland, where, as we shall afterwards see, epidemics of influenza have been often observed, the disease usually takes a course, according to the researches of Lange,[1] from north to south. On the other hand, in Iceland, as Finsen remarks,[2] influenza proceeds mostly from south to north; that there are, however, considerable exceptions to this rule is proved by the epidemic of 1843, in which various points on the island were almost simultaneously attacked,[3] and by that of the year 1856, in which the disease appeared first in the northern territory.[4]

The evidence is just as scanty for any regularity in the *length of time occupied in the extension of the epidemic* from country to country or from place to place. Not unfrequently, as for example, in the pandemics of 1833 and 1837, there have been large tracts of country smitten as if at a blow; in other cases, weeks and even months have passed before the disease extended to places even close at hand.

In the pandemic of 1826 on the continent of North America, the first cases appeared in Georgia during the month of January, and the disease was prevalent in Boston as early as the beginning of February. On the other hand, the pandemic of 1843 broke out first in New York in the middle of June, it appeared in the districts to the east of the lakes, as well as in the Central States, in July, and it was not until August that it appeared in the Southern States. In the epidemic of 1841, the disease prevailed in Prussia, Saxony, Hanover, and other states in January, while it showed itself first in Rhenish Prussia in April. In Greenland, it is usual for the beginning of the epidemic to appear in the northern

[1] 'Bemaerkninger om Grönlands Sygdomsforhold.' Kjöbenh. 1864, 13.
[2] 'Jagttagelser angaaende Sygdomsforholdene i Island.' Kjöbenh. 1874, 25.
[3] Hjaltelin, l. c.
[4] 'Sundhetscoll. Aarsberetning for 1856,' 66.

parts of the country from February to April, middle Greenland being visited by it in May and June, south Greenland in August or sometimes as late as September.

Finally, we have still to consider the circumstance, already adverted to, that there have been many remarkable exceptions to the incidence of influenza universally at a given place. Although it is the rule for the disease to extend uniformly to the whole population of a place, thereby stamping influenza with a peculiarity which belongs to no other infective disease except, perhaps, dengue, yet the exceptions are numerous and worthy of notice. Thus Hamilton (l. c.) records, for the epidemic of 1782 in Britain, that in several garrison towns, such as St. Albans, Aberdeen, and Dublin, the military part of the population was attacked first, and suffered most severely; in Novara also, according to the communications of Galli (l. c.), the disease, during the epidemic of 1833, showed itself first among the troops (in July), and not until a month later among the civil population; on the other hand, as Follet relates (l. c.), the epidemic of 1838 at St. Denis (Réunion), entirely spared the military garrisoning the place. Concerning the great epidemic of influenza which prevailed in upper India in 1832, Ludlow remarks:[1] "At Mhow it first attacked the natives in the Sudder Bazaar in considerable numbers; . . . it afterwards spread amongst the officers and servants. At a time when seventy or eighty men of the 65th Regiment were in hospital in consequence of the epidemic, not more than a case or two occurred in the 7th Cavalry, although both corps had lately arrived at the station." To the same effect, Mouat[2] reports from Bangalore: "Even here it had its anomalies, by affecting the Native Horse Artillery and entirely exempting the European Fort Artillery, who were about 100 strong." Regarding the epidemic at St. Petersburg in December, 1836, Seidlitz remarks:[3] "It did not attack the whole population of the capital at once, as in the former epidemics (1831 and 1833), but it appeared now here, now there, in many of the streets and in many of the households several weeks later than

[1] 'Calcutta Med. Trans.,' vi, 473.

[2] Ib., vii, 299.

[3] 'Hufeland's Journal der pract. Heilkde.,' 1837, lxxxv, 114.

in the rest." In the report of Staberoh for the Paris epidemic of 1837, it is stated :[1] "In this epidemic, just as in the sad time of the cholera, it was observed that particular parts of the city were spared without conceivable reason, while the neighbouring parts were severely attacked." In the Boston epidemic of 1825, according to the report of Dewees (l. c.), only the children sickened at its commencement (December), and it was not until the middle of January that adults were seized; also in the Dublin epidemic of 1847, it appears, from the narrative of Churchill (l. c.), to have been the children that suffered. In the epidemic of 1837 in Rennes, the malady had become widely distributed through the population as early as February, but, according to Toulmouche (l. c.), it was not until April that it appeared among the inmates of the central prison.

§ 4. Geographical Distribution.

The *geographical distribution of influenza* extends, without doubt, over the whole inhabited globe. From some large tracts of country, such as the West Coast of Africa, and the southern part of South America, no records of influenza epidemics have reached us, and the records for some other countries, such as the Cape, India, Australia and Polynesia, are but scanty; but this does not justify the conclusion that the disease has not been prevalent at all or only rarely in these countries, any more than the silence or the scattered notices of the chroniclers and physicians of the middle ages justify us in concluding that the disease was absent or of rare occurrence during that period. That influenza has been observed in several of the regions named, and especially in tropical latitudes, just as often as in Europe and in North America, we learn not only from the records of influenza epidemics as given in the chronological table, but also from the published observations of Bennet,[2] Ellis,[3] Wilkes,[4]

[1] Casper's 'Wochenschr. f. d. ges. Heilkde.,' 1837, 266.
[2] 'Lond. Med. Gaz.,' ix, 631.
[3] 'Polynesian Researches,' 1836, iii, 35.
[4] 'U. S. Exploring Expedition,' iii, 93.

Häole,[1] and Gulick[2] for Australia and Polynesia, of Don[3] and Lord[4] for India, of Rufz[5] and Carpentin[6] for the Antilles, as well of various authorities for Réunion and Mauritius. The predominance of the disease in temperate latitudes, it is quite obvious, is only an apparent one. The alleged *endemicity of influenza* also, in several countries situated within the cold zone, reduces itself, according to the data of Schleisner[7] Hjaltelin, and Finsen for Iceland, of Panum[8] for the Faröe Islands, and of Lange for Greenland, to the question whether influenza may not have been confused with the bronchial catarrhs that occur every year in wide distribution in these countries in spring and autumn, influenza itself being not really more frequent than in other latitudes. In Iceland, as Hjaltelin remarks, it appears at a time when it is prevalent elsewhere, either in Northern Europe, or in North America.

§ 5. Relation to Seasons and Weather.

Independent, therefore, of *climate*, as influenza appears to be in its geographical distribution, it is equally little influenced in its occurrence by *telluric conditions*. It has prevailed with as great intensity and in as great extent on marshy as upon dry soils, on impervious as upon porous soils, in valleys as on plateaus or in the hills, on the coast as in the interior, giving evidence, therein, of an ubiquity which belongs to no other of the acute infective diseases.

But influenza shows the same independence, as regards its origin, of the *seasons* and of the influences of the *weather;* and it is in that respect that it is marked off most essentially and most decidedly from epidemic bronchial catarrh.

Respecting the prevalence of influenza epidemics in par-

[1] 'Sandwich Island Notes,' Lond., 1854.
[2] L. c. ad ann. 1848.
[3] 'Bombay Med. Trans.,' iii, 10.
[4] 'Quart. Journ. of Cal. Med. Soc.,' i, 462.
[5] L. c., ad ann. 1850–51.
[6] 'Archiv de méd navale,' 1873, xx, 433.
[7] 'Island undersögt for et laegvidenskabel Synspunkt.' Kjöbenh. 1849, 41.
[8] 'Bibl. for Laeger,' 1847, i, 311.

ticular *seasons*, it is shown by the table of outbreaks given above, that of 125 epidemics or pandemics which ran their course independent of one another, fifty began in winter (December to February), thirty-five in spring (March to May), sixteen in summer (June to August), twenty-four in autumn (September to November). Certainly winter comes out very decidedly as the season of the year most favorable to the setting up of the disease; but we shall attach very limited importance to that as a factor in the pathogenesis when we call to mind that an epidemic once developed runs its course equally through all seasons of the year, of which fact the pandemics of 1580, 1781-82, 1831, 1832-33, 1836-37, are striking illustrations. The disease in Greenland exhibits the same behaviour in its progress; in that country it makes its appearance in the north during the winter, and it does not reach South Greenland usually before the summer (Lange). It is also noteworthy that the influenza season in several tropical regions—for example, the Indus valley (Lord), and the Antilles (Rufz)—is the hot season; of twenty-four epidemics of influenza observed in tropical latitudes, nine began in the hot season, seven in the cold, and seven in the transition period.

Just as influenza has prevailed in all seasons of the year, it has also occurred under the most various *conditions of the weather*—high and low temperature, steady and changeable weather, much or little atmospheric moisture. The disease has been very frequent in summer with a very high thermometer and great dryness of the air: for example, in Switzerland in 1557,[1] in the Rhine provinces and in Italy in 1580,[2] in the New England States in 1655,[3] at Augsburg in 1712, Nismes, Lille, Cusset and other places in France in 1762,[4] in Paris,[5] Northern Italy,[6] and Gibraltar[7] in 1782, at Plymouth and London in 1788, in the Antilles in 1823, in

[1] Gesner's explanation is (l. c.):—"I see no other cause than the southern character of almost the whole summer."

[2] Thomasius observes (l. c.):—"A certain accession became apparent in the hot weather."

[3] Webster, l. c.

[4] Razoux, Bouchet, Desbrest, l. c.

[5] Geoffroy, l. c.

[6] Rosa, l. c.

[7] Maclean (l. c. 291), speaking of the epidemic of influenza, adds the

China and at Manilla in 1830, at Stuttgart, Heidelberg, and in the Duchy of Nassau in 1831 and 1833. On the other hand, epidemics of influenza have been repeatedly observed during the cold weather of a severe and protracted winter: for example, in Northern Italy in 1709 and 1712, at Padua ("tempestate frigida et sicca, coelo die noctuque sereno," Morgagni, l. c.), in 1733 at Dijon and at York (where the epidemic broke out in the end of January during intense cold, the weather to the middle of the month having been moist and mild) in many parts of Germany during the very severe winter of 1742 (Juch), in the year 1775 in Clausenthal (where the disease appeared when a period of relaxing weather gave way to intense cold) in Reval and other parts of Russia in 1782, in 1827 in Siberia and the eastern parts of European Russia (where the sickness which had prevailed during severe cold disappeared on the setting in of moist and changeable weather), at Riga in 1832, in Würtemberg in 1837, at St. Petersburg and in Schleswig-Holstein in 1847, and in Central Franconia in 1857-58. That the prevalence of weather characterised by much atmospheric moisture and by heavy rains does not prevent epidemics of influenza from breaking out, is proved by the observations made in London in 1658, at Pressburg in 1675, at York in 1729, in the Netherlands, at Plymouth, and in Italy in 1732, in Yorkshire and at Boulogne in 1757, at Heidelberg in 1780, and to come to more recent times, at St. Petersburg in 1854-55. There is not the slightest ground for assuming a causal connexion between the production of influenza and certain states of the barometer. Just as little do later inquiries bear out the conjecture of Schönbein, and the statements of Spengler, Böckel, Jung, Granara, and others, that ozone has an influence on the development of epidemics of catarrh and influenza. As for the relation between sudden mists and the epidemics of influenza that have followed close upon them, some investigators have dwelt upon that sequence, but the theories of pathogenesis deduced therefrom may be relegated to that domain of fancies which the romancists of the profession have established in the province of etiology.

words:—"Which was attributed at that time to the extraordinary heat of the atmosphere."

§ 6. Special Liability of Natives and Exemption of Strangers.

Conditions of race are entirely without significance for the distribution of influenza. This is proved by observations made among mixed populations, by Chisholm, Rufz, Carpentin, and others for the Antilles, by Mouat for India, and by the chronicler of the epidemic of 1838 in Sydney. On the other hand, there have been noticed in various parts of the globe, certain remarkable differences between the *indigenous* and the *foreign* residents in their liability to influenza. The first information on that point occurs in Barclay's report on the epidemic of 1823 in the island of St. Thomas; "the most remarkable thing about this disease," says the report, "was that new arrivals almost without exception were exempted from it, while it was so generally distributed among acclimatised persons and natives that very few of them escaped. While the hospital was crowded with old and acclimatised soldiers, there was not a single case among the recruits who arrived in the beginning of the year (or six months after the outbreak of the epidemic). And the same circumstance was observed among the civil population; the disease prevailed most widely and most acutely among the coloured people, many of whom died, while not a single case of death happened, to my knowledge, among the whites." Still more markedly has this peculiarity in the incidence of the disease shown itself in Iceland and the Faröe Islands. "The influenza," says Schleisner (l. c. 43), "usually attacks the whole population (of Iceland), so that only a few persons escape it; but it is the rule for this disease, as it is for hydatids of the liver, to spare the Danes and other strangers. The medical officer for the district of Westerland reports that, in the epidemic of 1843, not one man fell ill on board the vessels manned by Danes, while in the craft manned by crews of Icelanders, not a single person escaped; and he adds that the same fact had been observed in the case of the Dutch and French fishermen in the epidemic of 1834." Regenburg[1] maintains that this assertion is unfounded; but Panum (l. c.) thinks his contradiction unjustified, all the

[1] 'Sundhedscoll. Forhandl. for Aaret 1848,' 13.

more so as the same observation—that natives and acclimatised persons alone sicken while strangers are spared—has been repeatedly made in the Faröe Islands. The earlier observations have also been confirmed by the experiences in the epidemic of 1856: "very few of the natives remained exempt from the disease, while no cases occurred, neither on this nor on former occasions, among the strangers and recent arrivals," says the above cited annual report. They are also borne out by the observations of Finsen[1] for the epidemics of 1862 and 1864; and Lange records the same fact for Greenland.[2]

The behaviour of influenza in the same part of the world, affords evidence of still another peculiarity, viz. that the disease is most apt to break out *on the arrival of ships from foreign ports*. One of the earliest recorded facts of the kind comes from the island of St. Kilda (Hebrides).[3] Panum, with reference to the same point in the Faröe Islands,[4] says: "It is a remarkable fact that the outbreak of these (influenza) epidemics stands in near connexion with the arrival of trading vessels, especially in the spring of the year; this circumstance cannot be regarded as merely accidental, inasmuch as the arrival of the first trader happens at various times, sometimes in March, sometimes in April, and sometimes not until May. We know besides, from the observations made by the government official Plöyem, during the seventeen years that he spent upon the island, that the epidemic broke out each time two or three days after the arrival of the ship, that the first cases of sickness were those of the factors of the cargo and the men in their employ, and that the disease afterwards spread over all Thorshaven and thence over the whole island." Finsen mentions the same fact for Iceland.[5] That there is something more than acci-

[1] L. c., 27. "De, som nylig ere komme fra Udlandet og som altsaa ikke ere akklimatiserede, synes kun undtagelsesvis at vaere modtagelige for denne Sygdom."

[2] L. c., 12. "Den europäiske Befolkning angribes i det Hele mindre hyppigt og mindre heftigt af denne Sygdom end de Indfödte; men Europäernes Modtagelighed for den tynes at stige, so längere de have opholt sig der i Landet, saa at Akklimatisernigen her virker paa en modsat Maade af, hvad der er Tilfaeldet ved de saedvanlige Klimatsygdomme."

[3] Gray, in 'Lond. Med. Communications,' 1724, i, 1.

[4] 'Bibl. for Laeger,' 1847, i, 312.

[5] L. c., p. 27.

dent in this, is proved by similar observations from other parts of the globe. Thus, in the Society Islands, according to the corroborative statements of Bennet and Ellis, influenza appears every time that a foreign ship arrives. For the Nicobars, Steen-Bille[1] mentions the outbreak of an influenza epidemic immediately on the arrival of the Danish corvette under his command; and Turner[2] states that the disease prevailed for the first time in the Navigator Islands in the year 1830, directly after the arrival of the ship which brought the missionaries. The fact itself can hardly be doubted; while the striking thing appears to me to be that the strangers themselves, in all the cases, have remained exempt or almost exempt from the epidemic.

§ 7. Influence of the Weather in the Causation.

The relations of influenza, so far as we know them, to climatic and telluric influences and to influences of the weather, do not help us much to explain the cause and origin of the malady, unless indeed we are content with conjectures that have no foundation and hypotheses for which there is no proof. The fact which has impressed the larger number of observers is that influenza has broken out and spread most frequently in the cold and moist weather which is especially apt to give rise to catarrhal sickness. On this observation has been based the assumption of an "evolution of influenza from bronchial catarrh," the disease being nothing else than a "catarrh of heightened potency prevailing as an epidemic." But observant and unprejudiced investigators have at no time failed to remark how groundless this theory is, and how independent influenza is of the state of the weather.

Thus, so early a writer as Salius Diversus[3] has pointed out with reference to the epidemic of 1580, that its origin could not be referred to any appreciable changes in the atmosphere,

[1] 'Bericht über die Reise der Corvette Galatea,' Kopenh, 1852, i, 244.
[2] L. c., ad ann. 1830.
[3] 'De febre pestilente tractatus,' Francof, 1586, 62.

inasmuch as the epidemic had occurred in various parts of Europe under different conditions of weather and at all seasons of the year; the cause of the disease, he thought, was to be conceived of rather as a "transmutatio in propriâ aeris substantiâ," and as a "levis corruptio." By the same kind of reasoning, Molineux arrived at a similar conclusion for the epidemic of 1693, which he was constrained to refer to something "subtle and occult" in the air. To the same effect are the statements of Whytt for the Edinburgh epidemic of 1757, of Baker[1] for the influenza in London in 1762, and of Fothergill for the disease in Northampton in 1775. Penada remarks that, in the epidemic in northern Italy in 1788, the sickness altogether spared the mountainous districts of the country, in which the influence of unfavourable weather must have been greatest and most persistent. Respecting the epidemic of 1800, Metzer[2] declares that he is obliged to dismiss all idea of a connexion between the origin of the disease and effects of the weather; and the same conclusion was arrived at by Barclay for St. Thomas in the Antilles (1823), by Ward for Penang (1831), by Lombard for the Geneva epidemic of the same year, by Berndt and Dieterich for the epidemic of 1833, by Greenhow and Graves for England and Ireland in 1837, and by others. Franque's *résumé* of the records of influenza epidemics during the years 1831-1855 is: "The manner of spreading, as well as the essential character of the disease, was the same in all these epidemics, whether they reached their height in the winter, spring, or summer months." Kollmann draws attention to the fact that in Java, in the year 1831, the disease attained the same distribution on the coasts exposed to the tropical heat, and in the mountainous parts of the interior subject to cold and wet or

[1] L. c., 8. "Enimvero si morbus, de quo in praesentia agitur, iis coeli proprietatibus, quae sensibus nostris se offerunt, ortum suum debuerit, qui, quaeso, factum est, ut non homines, loco proximi, eodem fere tempore aegrotaverint? Qui factum, ut morbus eos, quos millia non amplius II ab hac urbe disjungunt, serior longe, quam Londinenses ipsos corripuerit? Quidnam esse causae putemus, cur urbem Edinburgum ineunte Maio, aliquas vicinae Cambriae partes recedente tandem Junio nec prius invaserit? Profecto quidquid nobis de hac omni quaestione scire conceditur, angusta admodum metitur circumscriptio."

[2] L. c., 4.

variable weather. Finsen's conclusion, from his observations made in Iceland,[1] is that "states of the weather have no effect upon the origin of the infective disease in question, although they may affect its intensity." Some naval surgeons have thought themselves justified in referring the breaking out of the disease on board ship, especially on the high seas, to the effects of moist and cold weather on the crews; but the small reliance to be placed on this opinion is apparent from the observations of medical officers of the English navy during the prevalence of influenza in 1860 on board the ships of war cruising on the Australian station. "Where it originated," says the report,[2] "there is no means of ascertaining; it was generally ascribed to the state of the weather, but the influence of the weather as an exciting agent may be doubted, for the sickness seems to have been prevalent in all kinds of weather, whether cold or hot, wet or dry; it also attacked different ships' companies at different times, though the vessels lay close to one another." Another interesting contribution towards the same opinion occurs in the report, above mentioned, of Chaumezière, upon an outbreak of influenza on board a ship of war on a voyage from the West Coast of Africa to Brest: "The epidemic came upon us," we read,[3] "in the midst of the most favorable conditions for navigating, and with the temperature and other meteorological phenomena of a genial kind; it showed itself, as has so often happened, under the influence of a general cause, specific, but unknown in its essential nature, and independent of all appreciable climatic or meteorological conditions."

I have thought it necessary to discuss the foregoing question somewhat fully, inasmuch as even at the present day there are still many voices raised, and influential voices too, against the specific character of influenza, and in favour of its identity, both in etiology and in pathology, with epidemic bronchial catarrh.[4] I cannot conclude this section more suitably, perhaps, than by adducing the opinion arrived at by

[1] L. c., 26.

[2] 'Reports of the Navy for 1860,' p. 181.

[3] L. c., 37, 40.

[4] Thus, among others, Brochin in 'Dict. Encyclop. des sc. Med.,' 1872, xiii, Art. "Catarrh.," p. 242.

the Würtemberg physicians in the course of an investigation into the influenza epidemics of that kingdom in the years 1831-1858: "It appears from these researches," runs the report,[1] "that influenza prevailed sometimes in summer, sometimes in winter; sometimes in unusually warm weather, and sometimes in unusually cold; sometimes in dry weather, sometimes in wet. Considering, further, that the weather had thousands of times shown the same character as in influenza years, without influenza prevailing, and that influenza is usually prevalent at one and the same time over the whole of Europe and even in other parts of the world, where we may safely conclude that the weather had been of all kinds, *we are constrained to admit that influenza is altogether independent of weather conditions.* Should we desire, however, to open up the further question of an influence exerted by agencies or substances such as the electricity of the air, ozone and the like, we should be well advised to wait for further observations before taking the trouble to discuss it."

§ 8. A Specific Infection.

Influenza is a *specific infective disease* like cholera, typhoid, smallpox, and others, and it has at all times and in all places borne a stamp of uniformity in its configuration and in its course such as almost no other infective disease has. Its genesis presupposes, therefore, a *uniform and specific* cause, the origin and nature of which are still completely shrouded in obscurity. There can be no objection to calling this specific cause by the name of "miasma," so long as we remember that nothing more is expressed thereby than that which the physicians of the sixteenth and seventeenth centuries called a "fouling of the air," and that, in setting up a name in the place of an obscure conception, we do not bring ourselves by that means a single step nearer to a knowledge of the cause of the disease. All the opinions that have been put forward as to the nature of this "influenza miasma" are without any basis of fact; and that is true more especially of the theory, maintained as early as the eighteenth

[1] 'Württemb. med. Correspondenzbl.,' 1858, 188.

century,[1] and lately revived, of a "miasma vivum," or an organic (animal or vegetable) morbid poison, upon the carrying of which by the air the spread of the disease was thought to depend. But, as we have already seen, there is not the slightest cogent reason for supposing that the several parts of an influenza pandemic stand in a genetic relation to one another, or that it is a question of the conveyance of a disease-producing substance from place to place. We might with just as much probability assume that the cause of the disease has sprung up *de novo* at all places where its effects have been manifested, as that it has been distributed by the movement of the air. And, indeed, the circumstance that the progress of the disease does not depend on the direction of the wind, and may sometimes even go contrary to it, speaks in favour of the former view. Thus the epidemic in England in 1803 extended from south to north during the prevalence of north-easterly winds; and the report on the influenza epidemic of 1831-37 in Denmark says:[2] "Another interesting question is whether the disease went in a westerly direction with an easterly wind; Dr. Bremer, on comparing the direction of the wind for each day, came to the conclusion that there was not the smallest connexion discoverable between it and the spread of the epidemic, and that the course of the latter was wholly independent of the wind." In 1834, according to the account of Schleisner,[3] the influenza in Iceland went from north to south during a prolonged tract of westerly winds; and Finsen,[4] with reference to the distribution of the disease in general in that country, says: "An epidemic will spread over the whole island quite independent of wind and weather."

[1] "This disease," says Grant, in his account of the influenza epidemic of the year 1782, "is prevailing in certain districts of France at the moment of my present writing. It is there called 'la gripe,' from an insect of that name, which was very common in England and France during the past spring, and was supposed to have infected the air and imparted an injurious property to it. We know, however, that this view rests upon an error." According to Metzger ('Zur Geschichte der Frühlingsepidemie, &c.,' Königsb., 1782, note 5) a similar conjecture was made by Kant, that "the Russian trade with China had brought over some species of noxious insects, which might have got scattered abroad in course of time."

[2] 'Bibl. for Laeger,' 1847, July 6th.

[3] L. c. 44.

[4] L. c., 26.

§ 9. Alleged Contagiousness.

The question whether influenza is *communicable or contagious* has given occasion to a lively controversy. In more recent times the great majority of observers have answered it decidedly in the negative, not so much on the strength of the many single observations which tell against the communicability of the disease, as on the ground that the spread of influenza can be shown to have taken place quite independently of intercourse. To this argument I may add the fact that it has not spread more quickly in our own times, with their multiplied and perfected ways and means of communication, than in former decades or centuries. "The simple fact is to be recollected," says Jones,[1] "that this epidemic affects a whole region in the space of a week, nay, a whole continent as large as North America, together with all the West Indies, in the course of a few weeks, while the inhabitants could not within so short a time have had any communication or intercourse whatever across such a vast extent of country. This fact alone is sufficient to put all idea of its being propagated by contagion from one individual to another out of the question." The Provincial Medical Association in England gave special attention to the question of the contagiousness of influenza in the epidemic of 1836-37; the result of their observations thereon is summed up by Streeten in the following words:[2]—"The answers to this question—'Are you in possession of any proof of its having been communicated from one person to another'—are of an almost uniform tenour, the opinion of nearly all those who had the most extensive opportunities of investigating the disease, and the best means of arriving at a definite conclusion, being that there is no proof of the existence of any contagious principle by which it was propagated from one individual to another."

Partisans for the spread of influenza by contagion have found support for their views in the breaking out of the disease at various places, somewhat removed from the track

[1] 'Philadelphia Journ. of Med. and Phys. Sc.,' 1826, n. s., iv, 5.

[2] 'Trans. of the Prov. Med. and Surg. Assoc.,' 1838, vol. vi, pt. ii, 523.

of commerce, after the arrival of strangers; for example, the Danish physicians in Iceland and the Faröe Islands have found evidence of that kind in the outbreaks of influenza that have followed the arrival of foreign ships. Without questioning the accuracy of the observation itself, we may hesitate to accept the conclusions drawn from it when we duly keep in mind that the suspected importers of the morbid poison remain, as we are expressly told, unaffected by it, that they continue untouched by the epidemic, and, further, that the disease has not unfrequently appeared in these and other islands at the time of the ship's arrival although influenza had not been prevailing as an epidemic anywhere else, and most certainly not in those countries from which the ships had sailed. These considerations, taken along with peculiarities in the incidence and course of influenza epidemics—their occurrence suddenly and without prelude, and their attacking the people *en masse*, their equally sudden and complete extinction after a brief existence, generally of two to four weeks, and the frequent restriction of the disease to *one* place, while the whole country round has been completely free from it—all these points are so foreign to the mode of development and the mode of spreading proper to such maladies as originate beyond doubt through the communication of a morbid poison, that we shall find it hard to discover any reason for counting influenza among the contagious or communicable diseases.

§ 10. Uniformity of Type.

Few among the acute infective diseases have manifested, in their prevalence at all times and in all places, the stamp of uniformity so strongly *in the aggregate of symptoms* as influenza. The various epidemics, it is true, have differed much among themselves as regarded the character and course of the disease, but these differences—expressing themselves in a catarrhal affection particularly of the digestive mucous membrane, in the occurrence of exanthems, in the remarkably frequent accession of more severe inflammatory affections of the respiratory organs—have been found to be

associated either with a particular season of the year or kind of weather, or with something special in the locality, and have been not unfrequently determined more by the individuality of the sick person than by anything in the external causation.

§ 11. Coincident Outbreaks of Influenza among Horses.

I do not think that I need now enter at length upon a subject that was so keenly discussed some forty years ago, viz. *the relation of influenza epidemics to the epidemic prevalence of other infective diseases*, especially the cholera. That influenza should have preceded the outbreak of cholera in 1831, will be seen by a reference to the history of each of those diseases to have been accidental. If we may not speak of "accident" in the course of natural events, then, as Glnge justly remarks, we are at least debarred from calling it by any other name.

But I am not of opinion that there is any question of accident in *the relation of influenza epidemics to epizootics of the same character prevailing at the same time*, especially among horses, and, next to them, among dogs, cats, and the like. Even in the oldest epidemiological records, there are indications of these coincidences both as regards time and place, as well as of the identity or at least similarity of the form of disease; and the number of these observations is so remarkably large that the suggestion of an etiological and perhaps also pathological connexion between the epidemics, on the one hand, and those epizootics, on the other, may be regarded as provisionally proved, although it ought not at the same time to be left out of sight that the notion of "horse-influenza" has remained to the present day a somewhat vague one with veterinary surgeons, and that very various diseased processes appear to have been included therein.

The first communication relating to this matter which has any value occurs in the account by Molineux of the epidemic of 1693. "It was remarkable," says that writer, "that both in England and in Dublin shortly before the outbreak

of the influenza, there was a disease prevalent among the horses, not of a severe type but very general, which manifested itself chiefly in a discharge from the nostrils of the animals." To the same purport is the statement of Gibson[1] about an epizootic among horses which was rife in England and Scotland during the autumn and winter of 1732-33 at the time of the prevalence of influenza, and upon which Huxham also comments.[2] "About the end of the year 1732," says Gibson, "there was a very remarkable distemper among the horses in London and in several other parts of the kingdom. They were seized suddenly with a vehement, dry-sounding cough, which shook them so violently that some of them were often ready to drop down with hard straining and want of breath; their throats were raw and sore; . . . the running at the nose generally began the third day and continued in so profuse a manner for five or six days that some of them in that time discharged as much as two or three pails would hold of purulent matter, which, however, was generally of a laudable colour and good consistence. . . . "This distemper, *though in no ways mortal*, yet was so very catching that when any horse was seized with it I observed those that stood on each hand of him were generally infected as soon as he began to run at the nose, in the same manner as the smallpox communicates the infection when they are upon the turn." From the spring of the year 1767, in which influenza prevailed widely over Europe and North America, we have similar observations on the simultaneous occurrence of an epizootic in those countries among dogs and horses. "When I was in England in the year 1767," says Mumsen,[3] "we had cold north-easterly winds till late in May, following a severe winter. A pestilence broke out among the dogs and horses, ... it was called 'the horse cold' (*Pferde-schnupfen*); it occurred also among men, but it had no serious conse-

[1] 'Diseases of Horses.' Quoted by Heusinger, 'Rech. de pathol. comparée,' vol. ii, p. 220.

[2] 'Observat. de aere et morb. epidem. &c.,' Lond., 1752, 73—75.

[3] In a tract published anonymously, 'Kurze Nachricht von der Epid. Schnupfen-Krankheit, u. s. w.,' Hamburg, 1782, 20. There is another account of this epizootic in the 'Hannöv. Magazin,' 1767, p. 1645.

quences." Webster,[1] the American authority, mentions an epizootic during the same period among the horses in New England and New Jersey. In his account of the influenza epidemic of 1775 in England, Fothergill refers to the simultaneous occurrence of the disease among dogs and horses:—"The horses," he says, "had severe coughs, were hot, forbore eating, and were long in recovering;" and Parr adverts to the same fact in the English epidemic of 1782. Simmons[2] adduces the following observation communicated to him by Surgeon Boys, of the navy, for the epidemic of 1788:—"On the arrival of the Frigate 'Rose' at Portsmouth from Newfoundland (on the 4th of November, at the time when epidemic influenza was prevalent), all the dogs on board the vessel were seized with cough and catarrh, and shortly thereafter the whole ship's company sickened in a similar manner." During the influenza epidemic of 1837 in Cassel, "a catarrhal condition," as the report has it,[3] "showed itself among the horses." At the time when the disease was prevalent at the Cape of Good Hope in 1853 and 1854, the horses suffered from a similar malady, which destroyed many of them. In the report upon the influenza of 1857-58 in Würtemberg it is stated: "With reference to this point (influenza-like ailments among the domestic animals during the prevalence of influenza), the account by the Court Veterinary-Surgeon Wörz contains facts of interest. After similar ailments had manifested themselves in April and May, the disease spread in the end of December, in epidemic form, to a portion of the royal stud, in which were chiefly young horses, four and a half to six years old, partly of pure Arab breed, partly of Arab crossed with English; in a few days forty out of forty-four horses were seized, only four of the older horses escaping. Out of 100 horses in the royal stables, only thirteen were attacked up to the 12th of January, and these also were the younger horses, the severest case being in a thoroughbred Arab stallion. Thus only young horses, and of good strain, fell ill. The symptoms were exactly those of influenza in man.

[1] L. c., i, 256.
[2] L. c., 266.
[3] Casper, 'Wochenschr.,' 1837, 231.

. . . All the animals recovered." To conclude this summary of epizootics coinciding in time and place with epidemics of influenza, and probably to be accounted true cases of animal influenza, I shall advert further to the great horse plague which overran the North American continent as a pandemic in the years 1872 and 1873, at a time when influenza was rife over the whole of that continent. Hertwig[1] gives the following account of it from the reports of veterinary surgeons in America:—"The animals sickened always suddenly, with extreme weakness, coldness of the extremities, loss of appetite, redness of the conjunctiva, dry cough, and quick pulse. The respiration was impeded, the eyes ran, and there was a serous discharge from the nostrils, turning to a mucous consistence and a yellow colour about the third to the fifth day. Between the seventh and tenth days the cough became looser, the animals became more lively and inclined for their food, the pulse fell, and after ten to twelve days they were mostly well. In a few cases, which ran an especially rapid course, sweating set in on the third or fourth day; in other cases the illness was protracted over three weeks, and was followed by great prostration, and sometimes by general dropsy. The outbreak of the plague was sudden and general (as in the case of influenza), and the duration of it in the several localities averaged four to six weeks. On the whole, ninety per cent. of all the animals were attacked, and three to four per cent. of those attacked succumbed. The epidemic prevailed equally among horses of every age and race. At certain places, as, for example, Washington, the sickness was so widely spread that not one horse escaped it."

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CHAPTER II.

DENGUE.

§ 12. A Comparatively New Disease: its Symptoms.

Under the name *Dengue*,[1] the West Indian and United States physicians have described a peculiar disease which appeared as an epidemic in 1827–28 in the West Indies, on the seaboard of the Gulf of Mexico, and on the Atlantic coast of the Southern States of the Union; this disease attracted general attention both by the novelty of its form and by the enormous extent of its prevalence. Subsequent inquiries showed that the disease had occurred before at various parts of the globe. But it was not until the general outbreak of the disease in 1871–73 in the tropical regions of the Eastern Hemisphere that the learned world of medicine began to take much interest in it; and that is the period that gave rise to the excellent series of medical accounts which have furnished the desired information as to the peculiarities of the disease. Considering the small notice taken of dengue in German medical literature hitherto, I think it necessary to preface the account of the important points in its history, geography, and etiology, with a brief sketch of the disease itself.

[1] Men's wits have been taxed to find names for this disease. Thus the Spaniards called it *dengue* (corresponding in meaning to the French *minauderie*, and the English *dandy*) or *colorado;* the English and Americans have called it *break-bone* and *broken-wing*, the French *giraffe* and *bouquet* (whence the English corruption *bucket*), and the Brazilians *polka fever*. By the medical profession the disease has been named, according to the view taken of its nature, rheumatismus febrilis exanthematicus, scarlatina mitis, exanthesis arthrosia, and insolation fever; while some physicians have placed it beside febris remittens biliosa, or have professed to discover in it a mild form of yellow fever.

After an incubation stage, generally of short duration, averaging two to four days, and a prodomal stage (often wanting) characterised by a feeling of general weakness, pains in the head and joints, and gastric troubles, the outbreak of the disease is announced by chills, followed by a hot fit, in which the temperature rises very considerably in a few hours, not unfrequently reaching 40° C. (103·5° F.) and upwards. At the same time there occur extremely acute pains in the joints, at first usually in the smaller joints (fingers, hands, and feet), but afterwards in the larger as well (knee, elbow, shoulder) ; like the flitting pains of acute rheumatism, they often change their seat, and, in severe cases, they are so acute that the slightest pressure cannot be borne, and the patient is unable to make the least movement with the affected limb. As the disease develops in intensity, equally severe pains are felt in the bones and muscles both of the extremities and the trunk, sometimes also in the muscles of the eyeball with swelling of the skin and redness of the conjunctiva. The tongue is red or coated, and there is usually nausea and not rarely vomiting. The patient complains of acute pain in the head and back, and is restless and generally sleepless. Severe brain symptoms, such as delirium or a comatose state, are unusual ; more frequently observed are convulsions in young children, which are not, however, of serious import. In about one half of all the cases, an exanthem breaks out at this stage of the disease, in the form of a more or less uniform or spotted erythema which lasts only an hour or two. After this stage has lasted from twenty-four to forty-eight hours, a remission of the fever sets in and is rapidly completed, accompanied often with the outbreak of copious perspiration of a penetrating odour; this is followed by an abatement of the pains in the head, the joints, and the muscles, while the swelling disappears and much urine is passed, containing a large amount of urates. Shortly after there appears a second (terminal) exanthem (sometimes with febrile accompaniment), which is more constant than the former or initial one, and takes the form of bright red, slightly elevated, irregular spots, as in scarlet fever or measles, or it may simulate urticaria or pemphigus ; like the first exanthem, it has

often a duration as short as a few hours, only exceptionally lasting two to three days, and when it fades there is desquamation. With the outbreak of this exanthem, there often occurs swelling of the lymphatic glands of the neck, axilla, or groin; sometimes also angina, flow of saliva, or an aphthous affection of the mucous membrane of the mouth. These complications also are very soon over in mild cases which run a normal course, and the patient enters on the stage of convalescence after a period of sickness averaging six to seven days, being for the most part in a state of great weakness and anæmia; the convalescence is often very protracted in consequence of the long continuance of the joint affection, and under some circumstances it may go on for months.

Although the complications in this disease are troublesome, yet the prognosis is favourable even in comparatively severe cases. A fatal issue is extremely rare, and is for the most part observed only in children, the aged, or those persons who are attacked by dengue while suffering from chronic and severe organic disease.

The meagre post-mortem reports given by a few of the observers afford no satisfactory explanation of the nature of the disease. The changes found after death in the circulatory and respiratory apparatus are clearly due to other morbid conditions from which persons dying of dengue had suffered previously. Out of three cases in which a post-mortem examination was made, serous infiltration in the neighbourhood of some of the joints was found twice, and in the third case the crucial ligaments of the knee-joint were reddened.

§ 13. Geographical Distribution.

The earliest accounts[1] of the *epidemic prevalence of dengue*, and in fact of the occurrence of the disease at all, do not go farther back than the last twenty years of the preceding century, or beyond the years 1779 and 1780; in these years, if we may judge from isolated notices, the disease seems to have attained a considerable diffusion in the tropical and sub-

[1] All the authorities are given in the alphabetical synopsis of the literature of dengue at the end of the chapter.

tropical parts both of the Eastern and Western Hemispheres. "In the eleven hundred and ninety-third year of the Hegira," (*i.e.* 1779), says the chronicler Gaberti[1] "in the middle of the month Regeb, there appeared at Cairo and in the neighbourhood, a disease known as the knee-trouble (*abu rokab*); it threw all the people into a fever, not excepting even the little ones. Its first attack lasted for three days, after which the illness increased or diminished, according to the disposition of the individual. It was accompanied by pain in the joints, knees, and extremities, as well as inability to move, and often with swelling of the fingers. The after-pains lasted more than a month. The onset was sudden, the body being broken by it, and the head and knees taken hold of. The disease was cured through sweating, and by the use of the bath." According to the statements made to Ehrenberg (l. c.) by Dr. Marpurgo, the sickness was prevalent at the same time in Alexandria. For the same year there is an account of its prevalence in the month of March at Batavia by Bylon,[2] the medical officer of that town, who himself suffered from it, and for the year 1780 on the Coromandel coast by Persin, a missionary. "A fever of this kind occurred," he says, "on the Coromandel coast about the year 1780. Every one was attacked by it. The symptoms by which it was ushered in were almost the same as those premonitory of the plague—headache, lassitude, pains in the joints; but this epidemic had no bad consequences. The patients got rid of it in three days, under moderate diet and copious beverages."

There is another account for the year 1780 by Rush (l. c.) of an epidemic of "Bilious Remitting Fever" observed by him at Philadelphia in the months from July to October, and there can be no doubt that this was dengue fever. For the years 1784–88, there are accounts by Cubillas, Nieto de Pina, and Fernandez de Castilla of the prevalence of the same disease in Cadiz (1784 and 1788) and in Seville (1784 and 1785). The description given by these observers (Cubillas expressly mentions the exanthem, *una espulsion cutanea*

[1] Quoted by Pruner.

[2] In the 'Verhandel. van het Batav. Genootsch. der Konsten en Wetenschappen,' Deel ii, quoted by De Wilde, l. c., p. 426.

rosacea), referring as it does to the perspiration, with its pungent odour, which set in on the subsidence of the fever, and to the markedly favorable course of the disease in spite of symptoms often severe (pious people called it *piadosa* or the "merciful"), leaves no doubt as to the nature of the sickness. For the years following, down to 1824, there are only two accounts in medical writings which unquestionably refer to dengue. One of these is given by Leblond of an epidemic disease which prevailed at Santa Fé de Bogota, the capital of Granada, towards the end of last century (three years before his arrival there); it occurred at the commencement of the rains, and few of the inhabitants of the city escaped it. He describes it in the following words: "This malady began with headache, fever, and pains likes those of acute rheumatism, which led to its being mistaken at first for that disease; but, after a few hours in bed, the crisis showed itself in the groin and testis of the male, or in the groin and lumbar region of the female, with congestion and pain so severe as to draw loud cries from such of the patients as were over sensitive or had too little endurance. These congestions were soon dissipated by sweating." The second account comes from Lima, where, according to the statements of Pezet, the disease was so generally prevalent during the summer (January and February) of 1818, that out of 70,000 inhabitants in the city, only a few escaped having the sickness.[1]

There can be no doubt that in these, as well as in many other parts of the globe which we shall mention immediately, the disease has been much more common and more widely spread in past centuries and down to recent times, than we have any knowledge of from the scanty epidemiological records. Thus Cavell and Mouat state that in India dengue had occurred under the name of "three days' fever" long before 1824, and that when it appeared in that year, many elderly people easily recognised it as an old acquaintance. The accounts, also, of several medical authorities in Senegambia, Egypt, Arabia, and Oceania, speak of the disease as having occurred with comparative frequency in former times.

[1] This epidemic is mentioned (according to Smith) also by Paredes in the 'Heraldo de Lima,' 1854, No. 23–26.

Not until the great epidemic of dengue which overran the West Indies and part of the North and South American continents in 1827 and 1828, was the attention of physicians drawn to the peculiar characters of the disease; they learned to distinguish it from other diseases of the same class, with which it had often been confounded before, and thus the record of dengue, so far as historical research can make use of it, begins really with the third decade of the present century.

Coming to that period, we meet with the first great epidemic of dengue *in* 1824 *and* 1825 *in India and Further India.* The scanty information available about it (Kennedy, Mellis, Twining, Mouat, Cavell, Robinson, Voigt) hardly enables us to say definitely how far it spread or what course it took. It broke out in May, 1824, simultaneously, as would seem, in the province of Gujerat and at Rangoon, somewhat later it was at Chittagong, and in Calcutta during the rainy season (July and August), whence it spread up the river by Chinsura, Serampore and Chandernagore, as far as Beshampore. At the same time it was prevalent in Yanaon (mouth of the Godavery) and on the Coromandel coast (Madras and Pondicherry). In March, 1825 it reappeared in Beshampore, and extended thence during the rains along the valley of the Ganges to Channar, Patna, Ghazipore, Benares, and as far as Mirzapore. At the beginning of the cold season, the sickness disappeared at all these points, and so far as we learn from the data before us, it did not become general again until 1853 and 1854. Whether the epidemic of dengue observed by Ehrenberg at Suez, in December, 1824, had any connexion with the Indian one, cannot be ascertained in the absence of epidemiological records from Egypt for that period.

During the next thirty years dengue appears to have been epidemic in the Eastern Hemisphere at scattered points only, as in 1835 on the Arabian coast (*Pruner*); in June, July, and August, 1836, at Calcutta (*Raleigh*); at the same place in 1844 (*Goodeve* I); in August, 1845, at Cairo (*Pruner*); in 1847 at Cawnpore (*Goodeve* II); in 1845 and 1848 in the French possessions of Gorée and St. Louis in Senegambia (*Rey*); during the summer of 1851 in Réunion (*Dutroulau*, Ref. I, *Cotholendy*); and during the summer of 1852–53

in Tahiti (*Doutroulau*). In 1853 and 1854 dengue again became widely spread in Upper India, chiefly in the Bengal Presidency, as appears from the published accounts (*Goodeve* II, *Mackinnon*, *Sheriff*); according to Sheriff the disease was also prevalent at the time in Rangoon. This epidemic is succeeded by a series of local and circumscribed outbreaks as follow: in the French possessions in Senegambia from June to August, 1856 (*Doutroulau*, *Rey*), and in June and July, 1865 (Ref. I, *Thaly*); in 1856 at Benghazi, Tripoli (*Pasqua*), and in 1865, also in summer (July to September), in the Canary Islands (Ref. II, *Poggio*); further, in the summer (July to September) of 1867 at Cadiz (*Poggio*), and towards the end of the summer of 1868 at Port Said (*Vauvray*).

The history of dengue in the Eastern Hemisphere ends for the present with the great epidemic of 1870–73, in which the disease spread from the East African coast, over Arabia, Upper and Lower India, and as far as China. The first accounts of this outbreak date from Zanzibar, where the sickness appeared, according to Christie, in June, 1870, as a hitherto unknown malady, and continued until January, 1871, not sparing the coast territory adjoining (*Sheriff*). Next to that in the order of time comes the epidemic outbreak of dengue in the summer and autumn of 1871 at Aden (*Read*), Mecca, Medina, Jeddah, and other places on the Arabian coast (Ref. III, *Buez*, *Sheriff*); the disease became epidemic anew there in the spring of the following year, and in Jeddah again in 1873 (*Buez*). About the end of the summer of 1871, or shortly after it was at Aden, the sickness appeared at Port Said (*Vauvray*), and towards the end of the year (November) it broke out at Bombay (*Sheriff*) and almost simultaneously in a filthy quarter of Calcutta (*Raye*). From these points the disease spread with great rapidity, but the proper epidemic diffusion did not take place there until the hot season. As early as January isolated cases occurred in Cananore (*Sheriff*, Ref. V, *Fletcher*, *Sparrow*), and Calicut; in the city of Madras the outbreak began early in February (*Chipperfield*, *Sheriff*), cases were numerous in April, and the epidemic was at its height from August to October; in Calcutta the disease grew to an epidemic in March and raged

till June (Ref. VII, *Dunkley*); at Poonah and Secunderabad the first cases were in April; at Cochin, Quilon (*Morgan*), and Vellore in May, continuing in the last-named till October; and at Dacca (*Wise*) in June. The epidemic was at its height in Bengal from April to July (Ref. IV), and in the Presidencies of Madras (Ref. V) and Bombay (Ref. VI) from May to September; its appearance was later in the North-West Provinces of Allahabad, Oude, and Merut, where it prevailed from June to December, spreading as far as Umballa and Lodiana on the Punjaub frontier (Ref. IV). In the middle of April the sickness had reached Rangoon where it continued until July (*Slaughter, Burnett*) travelling to native Burmah somewhat later. It appeared at Shanghai in June, and at Amoy in August (*Müller* and *Manson*), whence it spread to places about; it reached the Island of Formosa (Ref. IX, *Galloway*) in October, and in December it came to Java, where it was prevalent until March, 1873 (*De Wilde*). In the year 1873, dengue was again observed in many places in Upper and Lower India, especially in the Madras Presidency, and from March to October in Cochin China (*Morice, Breton*). The last offshoots of this great epidemic were the outbreaks on the Islands of Mauritius and Réunion, which occurred respectively in January and February, 1873, and died out in May (*Brakenridge, Cotholendy*). Since the close of that outbreak the disease has been epidemic only once, as far as is known, in the Eastern Hemisphere—in 1878 at the port of Benghazi in Tripoli; according to the account of Pasqua the first cases occurred in the beginning of October; the disease assumed an epidemic character towards the end of the month, and spread through the greater part of the population, but it began to decline as early as the beginning of November, and by the end of that month it had died out completely.

In the Western Hemisphere, during the period subsequent to 1824, we meet first with an epidemic of dengue at Savannah, Ga., in the autumn (August to October) of 1826 (*Daniell, Waring*); this was followed by a long train of sickness in the next two years, extending over the whole West Indies and a great part of the Southern States of the Union, and not sparing even the northern parts of the South American coast.

This great epidemic started from three points: the Virgin Islands, St. Thomas, where it was prevalent from September, 1827, to January, 1828 (*Stedman, Lüders*), and Santa Cruz, where it broke out in October, 1827, and lasted till March, 1828 (*Stedman, Ruan*). From these centres it extended continuously in a southerly and westerly direction over the Lesser and Greater Antilles. Thus it prevailed on St. Bartholomew from November, 1827 until January, 1828 (*Cock*), on St Kitts from December to April (*Squaer*), on Antigua from January to March (*Nicholson, Furlonge*), on Gaudeloupe and Martinique from January to June (*Moreau de Jonnès*); on Barbadoes it broke out in March, and on Tabago, the most southern of the Lesser Antilles, it appeared in May, 1828. In the westerly direction it came to Jamaica in December, 1827 (*Maxwell, Stennet*), and to Cuba[1] in March, 1828 (*Tuite, Osgood*). On the Island of Curaçao, lying off the Venezuelan coast, the disease is said to have appeared as early as November, 1827; of its prevalence elsewhere in South America there are accounts only from the State of New Granada, from Carthagena (Ref. X), and from Bogota (*Waterson*). On the continent of North America the disease showed itself first at Pensacola, Flor., in the spring of 1828 (Ref. X); it appeared at Charleston (*Dickson* I) and New Orleans (*Dumarescq*) in June, at Savannah, Ga., in August (*Daniell, Waring*), and it reached Vera Cruz, Mexico, a little later (*Barrington*). Whether it attained a wider diffusion in Mexico is questionable. The Northern and Central States, as well as the interior of the United States, remained quite exempt from this epidemic, a few cases having been observed only at Philadelphia, among the crew of a vessel arrived from Havana (*Lehmann*). On the other hand, the disease was epidemic in the autumn of 1828 in the Bermudas (*Smart*).

For the next twenty years there are only a few trustworthy notices of epidemics of dengue in the Western Hemisphere.[2] These are for the Bermudas in 1837, for Rio de

[1] There is no information as to the occurrence of dengue in this epidemic on Porto Rico and Domingo.

[2] It seems to me to be very doubtful whether the disease observed by Hildreth ('Amer. Journ. of Med Sc., 1830, Feb., p. 330) at Marietta, Ohio,

Janeiro in 1845–1849 the disease having been prevalent firstly from December, 1845, to August, 1846 (*Lallemant* II), then from October, 1846, to March, 1847 (*Döllinger, Lallemant* I, II), and again during the summers of 1848 and 1849 (*Lallemant* II), and finally for New Orleans in the autumn of 1849 (*Fenner*).

In 1850 dengue again became widely diffused over the Southern States of the Union. It first appeared in July at Charleston (*Wragg, Dickson* II), in August simultaneously at Savannah (*Arnold* II), Augusta (*Campbell*) and New Orleans (*Fenner*); in September at Mobile (*Anderson* I), and Woodville, Miss. (*Holt*); and, finally, in October at Galveston, Texas, whence it spread along the coast by Matagorda and Lavacca as far as Fort Brown (*Jarvis*). The accounts that have come to hand of dengue in the Western Hemisphere since the subsidence of that outbreak point to more scattered epidemic occurrences of the disease. There was an outbreak at Callao and Lima (*Smith*) in the summer and autumn of 1851 (December to July), it prevailed at Mobile during the autumn of 1854 (*Ketchum*) as well as in Cuba and other West Indian Islands (*Arboleya*), in Martinique (*Ballot*), in the summer and autumn of 1860 (June to December) and in the Bermudas (*Smart*), where the disease recurred at the same time of the year in 1863. The latest information dates from 1873, and relates to epidemics of dengue in Alabama (*Anderson* II), at Vicksburg, Miss. (Ref. XI), and at Port Hudson and other places in Eastern Louisiana (*Marsh*).

There can be hardly any doubt that this survey of the epidemics of dengue hitherto known is not one that gives a complete outline of the diffusion of the disease in time and place, and that it is wanting in accurate information both as to the time of its first appearance at the various places, and as to the places where it has something of the character of an endemic malady. As regards the occurrence of dengue in Egypt, it was long ago stated by Ehrenberg that the disease was not rare there, and that it had been still more frequently observed in Arabia,

in the spring of 1829, was really dengue. I am unable to form an opinion as to the character of the epidemic which was prevalent, according to newspaper reports, at Iberville, La., in November, 1839.

especially in Jeddah and Jambo[1]; and this statement is confirmed for Arabia by Sheriff, and for Egypt by Vauvray, who remarks that dengue is common in that country at the time of the date-gathering, and is therefore known under the name of "fièvre des dattes." Rey mentions that in the period from 1847 to 1856, very few years passed without the disease showing itself in Tahiti, and that it had occurred also on board vessels at anchor in the harbour of Papité. In the Hawaiian Islands the malady appears to be not uncommon; the disease known to Duplouy under the name of "bonon" (sighs) as occurring there especially at the rainy season, may undoubtedly be set down as dengue. That it occurred in India before 1824 follows from the account of Cavell for Calcutta, where the disease used to be called "three-days' fever," and from the statement of Mouat that the disease had been observed at Beshampore on previous occasions, if not in such wide diffusion. Thaly remarks that it is prevalent in Senegambia, "sous le forme endémo-épidémique," and Verdier[2] also mentions its frequent occurrence there.[3] To the same effect is the statement of Smart, with reference to the Bermudas, that sporadic cases are observed every year there, and that the disease grows to an epidemic from time to time. It is said also by Hamilton to be a not uncommon malady in Honduras.

At other places, again, the disease appears to have occurred for the first time at a later date, or even quite recently, and to have been less frequently prevalent; thus, as Wise points out, it had never been observed prior to 1872 in the district of Dacca (Presidency of Bengal), nor, according to Sheriff, in the Presidency of Bombay, or in the City of Madras. The same is true for Amoy, according to Müller and Manson, and according to Christie for the East Coast of Africa, where the disease was quite unknown previous to the outbreak in 1871. In Réunion and Mauritius dengue became prevalent for the first time in 1851, according to

[1] He mentions that his travelling companion Hemprich had the dengue sickness on the Arabian coast.

[2] L. c., p. 53.

[3] It is a significant fact that the natives of Senegambia have a popular name for dengue, n'dagamonté or n'rogni.

the corroborative statements of Cotholendy and Brakenridge. This applies with even greater force to the outbreaks of the disease that have happened in recent times in the Western Hemisphere. Furlonge, Moreau de Jonnés, and almost all the chroniclers of the dengue epidemic of the West Indies in 1827 and 1828 declare that the disease had never occurred there previous to that time. The case is the same, according to Döllinger, for Brazil, where the disease showed itself for the first time in 1845 and 1846. In the United States, also, dengue is counted among the diseases that are only rarely met with.[1]

§ 14. Characteristics of Dengue as an Epidemic.

Before considering the question of the influence exerted upon the occurrence and diffusion of dengue by climate, weather, soil, and other factors in the environment of the individual, as well as by circumstances of race, age, and sex, I shall direct attention to some peculiarities in *the form and type* of its epidemics. Their sudden outbreak and their rapid diffusion, usually over the greater part of the population of the affected locality, afford a striking analogy with the phenomena observed in epidemics of influenza. Numerous authorities lay the greatest stress on these circumstances, which are not without significance in forming our opinion of the origin of the disease.

In the epidemic of dengue at Lima in 1818, only a few out of the whole 70,000 inhabitants of the city escaped having the sickness. In a postscript to Mouat's report of the Besham-pore epidemic of 1825, it is stated[2]: "At Churnarghur and in its immediate vicinity not fewer than 10,000 natives are said to have suffered from the disease at one period;" and Robinson says that hardly one man remained well in the

[1] In the 'Transact. of the Amer. Med. Assoc.,' 1851, iv. the epidemic of 1850 in the Southern States is referred to as follows: "This epidemic is rare in its occurrence. . . . . Since its former appearance in 1828 no analogous epidemic disease has prevailed to any great extent until the past summer."

[2] L. c., p. 49.

European regiment stationed at Ghazipoor during the prevalence of the same epidemic. In Stedman's report on the epidemic of 1827 in St. Thomas, we read: "The disease suddenly made its appearance in the island and attacked almost every individual in the town, which contains a population of about 12,000 souls. . . . The disease appeared suddenly and spread with rapidity. . . . Not a day passed but hundreds were attacked, and of this the consequence was a great interruption to trade." In Antigua, according to Nicholson, dengue prevailed to such an extent "that very few of the inhabitants of this town escaped its attack." Maxwell, for Jamaica,[1] speaks of the "rapid manner in which this singular . . . disease spread," and adds:—"There probably never was a more general epidemic than this. . . . Almost the whole white and coloured population were sooner or later affected, and very few remained who were not personally acquainted with the dandy fever." In his account of the epidemic in Martinique, Moreau de Jonnés says: "An official document affirms that one half of the inhabitants of Havana were attacked by it *almost simultaneously*, and it became necessary to erect temporary hospitals in several quarters of the city;" and Osgood caps this statement with the declaration that, in its further course, the epidemic extended to almost the whole population of the city. The "universality of the attack," and "its sudden appearance and rapid course," are specially emphasised as characteristic of the spread of the epidemic of 1828 in the Southern States of the Union.[2] Dickson begins his report upon this epidemic in Charleston with the words: "About the end of June, 1828, a singular disease made its appearance in our city, through which it spread with unexampled rapidity, soon bringing under its influence the greater part of our population." And Dumarescq writes to the same effect of the outbreak and progress of the sickness at that time in New Orleans. In Lallemant's (II) account of the epidemic of dengue at Rio de Janeiro in the summer of 1846–47, he says:[3]—"The quickness

[1] L. c., p. 151.

[2] 'Statist. Report on the Sickness and Mortality in the Army of the U.S. for the years 1821 to 1839,' Washington, 1840, p. 63.

[3] L. c., p. 506.

with which the disease spread was indeed most remarkable. . . . The sickness broke out almost simultaneously in the provincial capital of Raya Grande, opposite our capital, on the other side of the bay. On the *haciende*, in the neighbourhood, whole gangs of negroes fell ill, while in the various mercantile houses there were none of the principals and not always a half-crippled clerk to be found often for a whole week long. Ships were delayed in loading and unable to get to sea, and even the schools were deserted." Of the epidemic of 1851 in Réunion it is said:[1]—"It spread so rapidly that the hospitals were crowded in a few days;" and of the 1873 epidemic in the same colony, Cotholendy states that out of 35,000 inhabitants of the town of St. Denis there must have been some 20,000 taken ill. In Mauritius during that epidemic, according to Brackenridge, only a few of the population quite escaped the sickness. In the epidemic of 1871–72 at Calcutta, the number of cases was estimated at 75 per cent. of the population;[2] at many places in the Madras Presidency, where the disease was prevalent at the same time, scarcely one person remained well, and in the City of Madras hardly a house escaped (*Sheriff*). In Zanzibar, Christie says that almost the whole population sickened. In Rangoon (*Burnett*) at least two thirds of the inhabitants suffered in 1872, and the proportion was similar in the epidemic of the same year in Formosa (*Galloway*).

§ 15. A Disease of the Tropics and of Hot Seasons.

The area of diffusion of dengue, so far as it has become known to us hitherto, extends from 32° 47′ North latitude (Charleston in South Carolina, and Lodiana in India), to 23° 23′ S. (San Paulo in Brazil), unless, indeed, we stretch it so as to include the occasional outbreaks in Philadelphia (39° 56′ N.), and on the south coast of Spain (36° 10′ N.). The disease has therefore the character of a *highly tropical* malady, and in that respect, as well as in its inroads into

[1] Dutroulau, l. c., p. 89.

[2] 'Lancet,' 1872, June 8th, p. 811.

higher latitudes, it corresponds to yellow fever, although the latter extends in the Southern Hemisphere to 34° 46′ (Buenos Ayres).

The conclusion which may be drawn from this, that the origin of the disease depends upon atmospheric influences proper to the climate, particularly the temperature, finds support in the behaviour of the epidemic in regard to *seasons and weather*. Summer and the early autumn is the proper *dengue season*, as the dates already given will show. This is particularly true of those regions not strictly tropical, where the disease appears almost regularly in July and August, seldom so late as September, and always with unusually high temperature; even in the tropics most of the epidemics have happened in summer, or at least have attained their widest prevalence at that season. It is significant of the degree to which this association of dengue with certain seasonal influences has impressed observers, that many of them would make *weather characterised by high temperature* an essential condition for the prevalence of the disease.[1] But above all there is the consideration that a great fall in the temperature, or the setting in of absolutely cold weather, brings the epidemic of dengue to an end, just as it does in the case of yellow fever.

In the Philadelphia epidemic of 1780 the disease declined so much in the beginning of October, on the setting in of cold weather, with rain and rough east winds, that only a case here and there came under observation (Rush). Referring to the course of the sickness at Savannah in 1826, Waring says:[2] "During the winter of 1826-27, the break-bone

[1] Cotholendy (l. c., p. 194) gives the following interesting fact relating to this point from the Réunion epidemic of 1872:—"The colony possesses at Salazie a mineral spring situated at a height of about 900 metrès among the mountains occupying the centre of the island. Despite the considerable stir among travellers and sick people who left St. Denis to go to the spa during the epidemic, the disease at the latter attacked only two persons, who had acquired the germs of it at St. Denis. It had no power to propagate itself." The same thing was observed at Brûlé, situated 700 to 800 metrès above St. Denis. The author is doubtless right in referring the exemption of that place to the relatively cold climate proper to its elevated situation.

[2] L. c., p. 375.

fever has been suppressed by the frost," and of the epidemic of 1828 he says[1] that it "terminated under the effect of frost." The epidemic of 1872 at Madras ended in the middle of October after heavy rains and cold weather had set in (Sheriff); and, in like manner, in the epidemic of that year in the North-West Provinces, only occasional cases occurred from October onwards. In Savannah the sickness of 1850 died out in the same way on the setting in of frost; Arnold (II), who mentions that fact, sums up the experience derived from all places in the United States that had been visited by dengue: "This disease is undoubtedly affected by frost; the diminution of cases after a frost last fall was as marked as the diminution of cases in our endemic climate fever (*i.e.* yellow fever) usually is."

There has been an apparent exception to this in the occurrence of dengue during damp and cool weather in 1827-28 in several of the West India Islands, particularly St. Kitts, Antigua, St. Bartholomew, Jamaica, and Curaçao. "The weather," says Squaer,[2] "previous to the appearance under consideration, and during its continuance, was of a nature unprecedented in severity in the West Indies, at least for very many years. In the latter end of November, and nearly up to the present period (April, 1828), the weather became extremely boisterous, being nothing but a continuance of heavy rains and high winds; the evenings were cold, very cold for this country, so much so that we were obliged to shut our doors and windows on sitting down to dinner, and we found it requisite to cover ourselves with a blanket at night." Stennett's account for Jamaica is to the same effect, although it would appear that it was not there a matter of actually cold weather, but only of a relatively cool temperature, to which the inhabitants of those regions must have been particularly susceptible, for Stennett gives the morning reading of the thermometer (or the minimum) at or about 64° F. But at any rate the fact is important as bearing on the question how far the production of the disease is dependent on the warmth of the air.

The *amount of moisture in the air* is less decisive of the prevalence of dengue than the temperature, and is probably

[1] L. c., p. 391. [2] L. c., p. 25.

indeed, without significance; at least, the disease has broken out and run an epidemic course just as often during prolonged dryness of the air as during heavy rains. Examples of the latter are found in the epidemics of 1827–28 just mentioned, in several of the West India Islands, and further, in the epidemics in Réunion, Tahiti, Senegambia, and in that of 1860 in Martinique. On the other hand, the disease was prevalent at a time of absolute drought in 1780 at Philadelphia, in 1824 in Goojerat, in 1827 on St. Thomas—the first of the Antilles to be attacked on that occasion—in 1828 at Havana (*Tuite*), Savannah (*Waring*), Charleston (*Dickson* I), and Vera Cruz (*Stedman, Barrington*); further, in 1836 at Calcutta (*Raleigh*), in 1850 at Woodville, Tenn. (*Holt*), Augusta, Ga. (*Campbell*), Charleston (*Dickson* II), and other places in the United States,[1] and in 1872 at Madras (*Sheriff*).

§ 16. Influence of Locality.

Notwithstanding the lively interest which the disease has excited among medical observers at all the places where it occurs, the question of the influence exerted by *circumstances of locality* on the production of the morbid condition has been little adverted to; it has not been discussed from any side with the thoroughness to be wished, and consequently the information to be got from the epidemiological records before us is too scanty to suffice for even an approximate solution of this question.

The first noteworthy circumstance is that the area of distribution of the disease in the Western Hemisphere has been almost limited to *places on the coast*. The data before us on this point from the West Indian epidemic of 1827-28 are very precise. "The dengue," says Osgood, "has not spread into the interior of Cuba, although at the end of five months from the time of its rise in Havana, it con-

[1] In the General Report (xii) on the dengue epidemic of 1850 in the Southern States it is stated: "Long-continued dry and hot weather preceded the development of the epidemic in all the places in which it has been described."

tinues to attack most of the persons who come to the city from the country or from any place (of the interior) where it has not prevailed." Stennet makes a similar observation for Jamaica: "It has passed all over the island, chiefly, however, prevailing in the towns along the sea shore." Also in the later epidemics in the West Indies—in Cuba, Martinique and other islands—it is always the occurrence of the disease in the coast towns that is spoken of, and there is never any mention of its further epidemic extension into the interior. This fact in the distribution of the disease comes out also very prominently in the Southern States of the Union, where dengue has been prevalent almost exclusively on the coast, although it spreads along the Mississippi as far up as Vicksburg. In Brazil and Peru also, so far as we know, it has been strictly limited to the coast. The same is true of the outbreaks of dengue on the east and west coasts of Africa, on the coast of Arabia, and elsewhere; and even in the isolated outbreaks of the disease in Spain, it has always been a few coast places that have suffered, without transmitting the sickness into the interior.

A second fact showing the influence of locality on the production and diffusion of dengue is found in the limitation of the disease, as an epidemic, for the most part to towns; in which respect also it has an affinity with yellow fever. In the report (XII) on the 1850 epidemic in the United States, it is expressly mentioned that, excepting in New Iberia, La., towns only were affected; in the West Indies also, as we have seen, it was almost exclusively in towns situated on the coast that the disease occurred in epidemic form. I reserve other observations relating to the point until I come to the question of the communicability of dengue. A natural explanation of the fact readily suggests itself in the cramped and crowded life of urban populations and the hygienic abuses inevitably associated therewith, presenting a particularly favorable soil for this as for other morbid poisons to develop in. De Wilde, in his account of the dengue epidemic that occurred in 1872 among the garrison of Fort Willem I in Java, points out that the disease mostly attacked the occupants of two block-houses occupying a particularly unhealthy site, and attacked them, too, without distinction of race,

rank, sex, or age; those individuals, on the other hand, who were housed under more favorable conditions, enjoyed a striking exemption; and he adds: "There need be no hesitation in assuming that the unhealthiness of the houses was the immediate cause of their selection." In the Calcutta epidemic of 1871-72, the disease was first developed among the Jews occupying the poorest and most densely populated parts of the city (Ref. VII). Smart is of opinion that crowded rooms and the 'tween decks of ships are the favourite seats of the disease, and the most intense foci of sickness. In the Philadelphia epidemic of 1780, dengue was most prevalent in the streets along the Delaware shore, filthy, overcrowded, and occupied by the poor, while those situated at some distance from the river were only slightly affected. At St. Denis (Réunion), according to Cctholendy, the overpopulated parts of the town, with the houses crowded close together, were the chief seat of the disease, while those houses of the town proper, situated within gardens, and therefore separated from the adjoining buildings, suffered from the sickness only here and there, but they suffered throughout the whole household when once attacked. In many other instances, however, the epidemic has spread uniformly over the whole of a town.

§ 17. Slight Influence of Race, etc.

An almost absolute independence of the circumstances of *race, nationality, age, and sex* may be recognised in the distribution of the disease at all times and in every place. Only a few observers, such as Squaer, Maxwell, and Stedman in the West Indian epidemic of 1827, have found that the negroes had the sickness more rarely or more mildly. In the Java epidemic of 1872, according to De Wilde, that race enjoyed an almost complete immunity. In like manner Pasqua says of the epidemic at Benghazi: "It was notable for the remarkable immunity experienced by the blacks;" but he adds that this race at the same time had no exemption in Egypt, Senegal, and elsewhere. Christie, also, is of opinion that the natives of Zanzibar suffered less than the

Europeans. On the other hand, Kennedy, in Goojerat, 1824, and Manson and Müller at Amoy, 1872, met with more numerous and more severe cases among the natives than among the Europeans. In Cochin China, according to Breton, cases of sickness were much more frequent among the Anamese part of the population than among the Chinese. Wise, in the Dacca epidemic of 1872, appears to have found a peculiar predisposition to dengue sickness in persons suffering from surgical complaints. According to Brackenridge, children enjoyed a certain immunity in the Mauritius epidemic of 1873. It must remain an open question how far all these isolated data may be trusted; at all events, differences of that kind in the liability to sickness in a mixed population are only rare exceptions to the general rule above stated.

§ 18. Specific Character: Question of its Communicability.

As to *the specific nature of dengue* there can be no reasonable doubt. All observers take the malady to be an infective disease (although they may express this idea in different ways), with a specific cause, underlying which is a morbid poison. The origin and nature of this poison[1] is enveloped, however, in the same obscurity that hangs around the virus of every other acute infective disease; we can only say this much of it with certainty, that its activity (perhaps also its production) is materially under the influence of high temperature, that its manifestations point to a certain connexion with the sea coasts and the shores of great rivers, and that it appears to attain its widest diffusion where the population is densest. Whether this poison springs up *de novo* at all points where its potency is manifested; whether it is endemic only at a few places and spreads from these under favouring circumstances, so giving rise to the more general prevalence of the

[1] This is another of those diseases in which there has naturally been no lack of conjecture as to the parasitic nature of the morbid poison. Thus, Dickson (II) and Poggio make it out to be a living germ; and Charles, on examining the blood of dengue patients, found therein, especially from the third to the sixth day, small round corpuscles which he takes to be organic elements, and to which he appears disposed to assign some specific importance in the morbid process.

disease; what are the ways or the media by which this diffusion is effected—all these questions and others connected with them cannot be answered with certainty in the present state of our knowledge. At the first general outbreak of dengue in India in 1824, and in the West Indies and United States in 1827-28, the belief in the communicability or contagiousness of the disease found only a few adherents.[1] During the next forty years, while the disease was getting more and more limited in its area and becoming all the more adapted therefore to furnish the data for a conclusion as to its true mode of spreading, there were still only a few observers who pronounced decidedly for its contagious character. It was not until the great dengue epidemic of 1871-73, in the tropical regions of the Eastern Hemisphere, that a change of opinion was effected; the position of the anti-contagionists was practically abandoned, and the disease was recognised to be "in the highest degree contagious."

The proof that the disease originates by way of communication rests on the fact of its having been observed to break out directly after sick persons from an infected locality have arrived at places that had been up to that time healthy; so that the diffusion of the disease could be traced, more especially in the epidemic of 1871–73 in the East, from port to port, and from country to country, along the highways of land or water traffic, as if step by step. The affirmative answer on the question of contagiousness derives additional support from the often observed fact, that the outbreak of the sickness takes place in the immediate

[1] Among the physicians in India who observed the 1824 epidemic and the subsequent isolated outbreaks of the disease at Calcutta, Cawnpore, and other places, there was not one who declared for its contagiousness; so that Jackson is right in saying (l. c., p. 208): "It is strange that in the epidemic of 1824 in Calcutta [and other parts of India] almost all the medical men of the day believed that the disease was not communicated from man to man, whilst the reverse is the case in the late epidemic (1871-72), the supporters of the non-contagious theory being in a very decided minority." Among the West Indian physicians only Stedman and Cock came forward as decided adherents of the theory of contagiousness, and Dickson was its only supporter in the United States. In 1850, Dickson still remained true to that opinion, as against Wragg, Arnold, and various others who pronounced decidedly against the contagiousness of dengue.

vicinity of the first patients, that new foci of sickness form around that point, the disease thus spreading through families, houses, and streets, that those persons who have come oftenest into direct contact with the sick (such as relatives, medical attendants, and nurses) become victims of the epidemic most surely and most speedily, and that isolation of the sick or avoidance of intercourse with them ensures protection from the disease.

However striking many of the accepted observations in favour of the communicability of dengue may seem at the first glance, we should not be justified, on an impartial examination of them, in taking them all as equally good evidence; and if criticism can of itself shake the belief in the "eminent contagiousness" of the disease, there are additional facts, overlooked by the partisans of the doctrine of contagiousness, which should weigh in the scale no less. I shall limit myself to a discussion of the more important of these facts, after first directing attention to the absence of critical caution in tracing the outbreaks at particular localities to the arrival of ships or to movements on land, and therein finding the explanation.

While Cock[1] considered it proved that the sickness of 1827 was imported into St. Kitts by a ship from St. Bartholomew, Squaer, who was an eye witness of the epidemic on the island, says:[2] "It is not meant that the present disease should be considered as having been brought to this island by means of communication." Waterson also[3] speaks decidedly in favour of the *de novo* origin of the disease on that occasion. The outbreak of the disease at Havana in 1828 was traced to the arrival of a Spanish flotilla which came from the South American coast, having touched at Curaçao where dengue was epidemic; but Robert observes:[4] "It is right, however, to state that before the arrival of Admiral Laborde at Havana, there was a disease almost of the same nature in the port of Santiago de Cuba, where the crew and troops on board the Spanish frigate 'Aretuza' were attacked." In the account of the epidemic of 1850 in New Iberia, La., the outbreak was traced to a person who came from New Orleans and took the disease shortly after he arrived; "but,"

[1] L. c., p. 46. [2] L. c., p. 22. [3] L. c., p. 309. [4] L. c., p. 315.

says the chronicler, "there was no connexion between the first and second cases, the subject of the first being a stranger, whom no one but his medical attendant had visited. . . . Again, persons from the country who visited our village and remained any length of time on business or to nurse the sick, took the disease on their return home, but in no instance did it spread among the other members of the family." Vauvray thought that the dengue epidemic of 1871 at Port Said should be attributed to importation from Aden, but how far he was right will appear from the words added by himself: "I ought to mention, at the same time, that the dengue fever was epidemic in 1868, and according to my civilian colleagues there have been some sporadic cases of it every year towards autumn. If it be an endemic malady, it must still be admitted that at certain junctures it takes on an epidemic character." The outbreak at Lima in 1852 was thought to be connected either with importation of Chinese or with German immigrants from Rio. The former assumption is said by Smith, who has written on the subject, to be quite untenable; the latter he looks on as better grounded, but the description which he gives of the disease observed by himself among the Germans, shows that these unfortunates had suffered not from dengue but from severe forms of typhus.[1]

Nothing, in my opinion, tells so little in favour of, and so much against contagiousness, as the sudden and simultaneous appearance of the disease over a great part of the population, an observation that has been made at the most diverse points of the wide area covered by dengue. The facts already mentioned at the beginning of this inquiry are borne out by others of the same kind: "In New Orleans," says Dumarescq referring to the epidemic of 1828, "the disease was not propagated by contagion; four persons were attacked by it at the same time, and its spread was so rapid among the inhabitants that

[1] [A perusal of Dr. Smith's papers bears out the impression of Professor Hirsch, stated elsewhere, that it was not yellow fever that the German immigrants suffered from. At the same time the symptoms described by Dr. Smith correspond closely to those of dengue. The assertion that the ships conveying the emigrants from Bremen had touched at Rio on the way to Peru has not been substantiated. See Smith, 'Epidemiol. Trans.,' i, p. 286.]

in eight or ten days at least one third of the population was labouring under its influence." Twining says of the spread of the disease at Calcutta in 1824: "I do not know that any proof can be adduced of the contagious nature of this disease: on the contrary, I believe it was not communicable from one person to another, because it arose at the same time in remote parts of the town, and affected persons who had not had any communication with sick people. Its progress was not that slow and gradual march which depends on personal communication and can often be traced." Wragg thus sums up his experiences of the Charleston epidemic of 1850, which completely agree with the observations made there by Dickson in the epidemic of 1828 (see p. 67): "I do not think it contagious, because its invasion was so sudden and general all over the city, that any attempt to trace it from patient to patient, from house to house, or from quarter to quarter would utterly fail."

In this peculiarity of its epidemic behaviour, dengue connects in the most obvious way with influenza; and if the facts concerning influenza are such as to furnish a real argument against its contagiousness, we may take up the same critical attitude towards dengue. A second analogy between those two diseases lies in the fact that at the time of dengue epidemics, just as during epidemics of influenza, there have been noticed cases of animal sickness with more or less distinctly typical marks of dengue. Observations of that kind are reported by Cubillas from the epidemic of 1784 at Cadiz, by Martialis, based on reports in the Indian papers ('Friend of India,' &c.) from the 1872 epidemic at Baroda, where it was chiefly the cattle that suffered, and by Slaughter for the epidemic of the same year at Rangoon, where the victims were mostly dogs and cats.

There is a third argument against the "eminently contagious" character of dengue, namely, the somewhat narrow limits of the epidemic as observed in many instances, or the association of it with definite local conditions, beyond which the disease has not penetrated notwithstanding importations on a large scale. Waring concludes his description of the Savannah epidemic of 1828 with the remark: "Not one case is known to have originated upon the plantations, and

although some persons, after having imbibed them [the germs], went into the country, it is not ascertained that they propagated them in a single instance. These facts conclusively finish the argument, it appears to me, against the suspicion of contagion or importation." To the same effect is Osgood's opinion, as we have seen, on the immunity enjoyed by the interior of Cuba while dengue was prevalent at Havana and other coast places, and despite the conveyance inland of many cases of the disease. Against contagiousness, Wragg adduces as evidence the fact that the epidemic of 1850 was confined to the immediate neighbourhood of Charleston, and did not reach the interior, "although the city was thronged with persons from all parts of the neighbouring country on business. . . . . There are several instances in which, after the greatest possible exposure, the disease was not developed." Pasqua also states that the epidemic of 1878 at Benghazi was not imported but had an autochthonous origin there, and that it was strictly confined to the town, not a single case having occurred outside the walls.

In view of these opposing facts, we shall have to treat the question of the communicability of dengue for the present as an open one. And it will be equally necessary to withhold an opinion as to *where the disease is indigenous* and where it is merely introduced. This caution of criticism is all the more required, inasmuch as the area of diffusion of the sickness up to the present, as well as the epidemiological history, have been made known to us only in scanty outlines. If, in forming an opinion as to endemicity, we were to assume that every place within the dengue area was an endemic focus because the disease had broken out there once or oftener when there was no proof of importation, then there would be hardly any locality left to exclude from the list of endemic centres.

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CHAPTER III.

SWEATING SICKNESS AND MILIARY FEVER.

A. *The English Sweating Sickness.*[1]

§ 19. Historical Outbreaks: the Symptoms.

In August, 1486, shortly before the battle of Bosworth Field, won by the army of Henry VII, a destructive disease broke out among the troops, to which the name of *sweating sickness* was given, from its most important symptom. It spread rapidly over the whole country, carrying off many victims wherever it appeared, and it died out towards the end of the year, probably in November. The same pestilence broke out anew in London in the summer of 1507, but this time its diffusion was less extensive, and it had disappeared before the end of autumn. A third outbreak of the sweating sickness took place in July, 1518. On that occasion also the epidemic began in London, and it spread over a great part of England within the next six months; it is reported, but on somewhat unreliable evidence, to have been even prevalent at Calais among the English living there. In all the epidemics up to this time, not counting the occurrence at Calais just mentioned, the disease had been confined exclusively to England (Scotland and Ireland being completely exempt), but on its fourth outbreak, in May, 1529, it spread over a large part of the Continent of Europe. This time also the sickness began in London, and having

[1] A full list of all the accounts of the English sweating sickness given by contemporary physicians or chroniclers will be found in the 'Scriptores de Sudore Anglico superstites, &c.,' Jena, 1847, drawn up by Gruner, and completed and published by Häser. There is a detailed history of the sickness, also with a complete list of authorities, among the historical and pathological writings of Hecker, edited by me, under the title "Die grossen Volkskrankheiten des Mittelalters," Berlin, 1865.

spread with great rapidity over the whole of England to the Scottish border, which it did not cross, it appeared in July in the Baltic and North Sea ports of Germany, and within the next five months it had extended from them over the whole of Germany, as well as through the Netherlands, Denmark, Sweden, Livonia, Lithuania, Russia, and Poland. In Switzerland, where the sickness occurred first in December, it was limited to the northern part (Basel, Solothurn, and Bern), while France and the countries of southern Europe escaped it altogether. Another twenty-five years passed by without a single trace of this malady showing itself anywhere, when in April, 1551, it broke out anew in England, this time at Shrewsbury, and overran the whole country with great virulence as far as the Scottish border, dying out in September. The Continent of Europe, as well as Scotland and Ireland, into which the pestilence had never penetrated, remained quite unaffected by this epidemic, although, according to a very doubtful rumour, cases of sweating sickness occurred at the same time among the English living in foreign countries such as France, the Netherlands, and Spain.

The descriptions which contemporary writers have left of this disease, afterwards known by the name of "the English sweat," have in general a stamp of uniformity, although it is plain that the type must have been modified by local influences, particularly the kind of treatment followed, which was sometimes rational and to the purpose, at other times injurious.

There were rarely any prodromal symptoms to an outbreak of the disease; it mostly set in abruptly, and usually in the night time, with chills followed by heats: the patients complained at the same time of oppressive palpitations, headache, want of breath, a sense of pressure or tightness in the region of the stomach, and sickness. As these symptoms rose in intensity, along with general turgescence of the skin, a profuse sweat broke out over the whole body, accompanied, as some observers mention, by a spotted, papular or vesicular exanthem.[1] In

[1] Castricus, one of the most reliable authorities, who made his observations at Antwerp, compares the exanthem to the maculæ of measles; Tyengius, another Dutch physician, speaks of "pustulæ parvæ exasperantes;" and an English observer, Stapleton, alludes to them as "maculæ quas rhonheas [probably measles] vocant."

cases running a favorable course, the morbid phenomena declined after twenty-four or forty-eight hours, the sweats became gradually less, an abundant flow of urine took place, the skin desquamated, and after a week or two the recovery was complete. A grave type of the malady was indicated at the outset by severe cerebral symptoms, intense headache, delirium, convulsions, and a quickly developed comatose condition (*somnolentia et inevitabilis sopor*), in which it is said the patients invariably died unless they were aroused out of it. Among other serious symptoms mentioned, were colliquative sweating and extreme scantiness of breath; death would then occur with symptoms of dyspnœa or general paralysis, sometimes only a few hours from the beginning of the illness. One or more relapses were common, especially if the patient had sweated little in the first attack.

The duration of the epidemic was at some places only a few days (three to seven), it was mostly limited to one or two weeks, and it was the exception for it to last several weeks. Despite this relatively short continuance of the sickness, the number of cases was enormous; the mortality also was at many places very great, while at other places only a few died out of all the thousands that fell ill.[1] It is difficult at the present day to decide how far local circumstances, particularly the mode of treatment, may have influenced the deathrate; at all events, the diaphoretic method, which was part of the therapeutic notions of the time, contributed materially to the high mortality. It was not until the later epidemics that the English physicians became convinced of the injuriousness of that procedure; they confined themselves to a more expectant treatment, so that both in England and in Germany (where the rational practice did not become current until near the end of the epidemic) the sickness began to lose much of its terrible character, and the mortality to fall to a minimum.

[1] Without venturing to trust implicitly to the data as to the number of deaths from sweating sickness at many of the localities, we should be justified, from a certain agreement among them, in concluding that the type of the disease had been disastrous in some places and very mild in others. Thus, more than 1000 persons are said to have died of it at Hamburg; at Freiberg (Saxony), 300, some say 600; at Copenhagen the mortality on some days appears to have reached the enormous figure of 400; at Augsburg there were 18,000 cases, with 1400 deaths; in Antwerp the dead were counted to the number of 300 or 400 within a few days. On the other hand, out of 4000 cases at Stuttgart only 6 died, of 50 patients at Marburg only 1 died, while at Strasburg and other places in Alsace the deathrate was at a minimum, notwithstanding the enormous diffusion of the malady.

§ 20. Associated Circumstances.

As regards etiology, those who observed the pestilence in England brought forward the fact that its appearance on every occasion was preceded by heavy rains, and in some places by inundations; on the Continent of Europe also, the epidemic in the summer of 1529 was ushered in by the same kind of weather and by widespread inundations. It was always in spring or summer that the disease appeared, and on no occasion did it remain longer than the beginning of winter. Communication of the disease through contagion is decidedly negatived by most of the observers, and these the most trustworthy, and we may therefore set aside the assertion of a few chroniclers that the sickness was carried in 1529 by ships from England to Hamburg.

Finally, it should be mentioned that those in the prime of life and of the male sex suffered most; children and the aged, if attacked at all, suffered less severely, and at some places the indigent part of the population enjoyed a striking immunity from the disease.[1]

Thus the "English sweat," with its five outbreaks within the period from 1486 to 1551, forms a completed episode in the history of pestilence. Just as it appeared suddenly in 1486 as a malady quite unknown to the doctors or to the public, a hitherto unheard of phenomenon, so in 1551 it went clean away from the earth and from men's memories, leaving no trace. Not until two hundred years after do we again meet with epidemic outbreaks of a kind of sweating sickness, which, if not identical with the English sweat, is still nearly related to it in every respect, whether superficial or essential. The history of it is sketched in its main outlines in the section that follows.

[1] Thus Kock and others report from Lubeck: "The poor people and those living in cellars or sleeping on the ground were free from the sickness," and Renner, for Bremen, says: "The sweating sickness went most among the rich people."

B. *Miliary Fever.* (*Suette des Picards. Suette miliaire.*)

§ 21. Confused Identity in Old Writings.

In the middle of the seventeenth century attention was drawn in several parts of Germany, first at Leipzig and afterwards at Hamburg, Augsburg, and other places, to a severe disease of puerperal women, which had not been observed before, or not at least distinctively recognised; from its predominant symptom, an exanthem, observers gave it the name of *Friesel*[1] (the purples), *febris miliaris*, or *purpura*. The exanthem was a lighter or darker shade of red, more or less uniformly spread over the whole skin, upon which millet-seed nodules (papules) or small blebs filled with serum (miliaria), often formed. The exanthem caused much itching, remained for a few days at its height, then faded, and was followed by desquamation of the skin. In the larger number of cases, the disease ran a fatal course, with symptoms of great disturbance, such as high fever, quick pulse, want of breath, extreme restlessness, delirium, bleeding from the nose, and convulsions; and not unfrequently death occurred even before the rash came out.

It must remain an unsettled question which of the diseases now known to us this "childbed purples" corresponded to. Probably it was scarlet fever, which is well known to occur often in puerperal women, and to run usually a very unfavorable course. Moreover, at the period from which these reports come down to us, scarlet fever was not much known as a special form of disease, being hardly distinguished from other exanthematous diseases, especially from measles. There is the less likelihood of coming to a certain decision, for the reason that shortly after the publication of the first observations by Hoppe[2] and Gottfried Welsch,[3] numerous errors crept into the doctrine of "the purples;" the exanthem, of all the morbid phenomena, was selected for notice

[1] The name is derived from the rough and reddened surface of the skin, suggesting frieze.

[2] 'De purpura dissertatio,' Lips., 1652.

[3] 'Historia medica novi istius puerperarum morbi, qui ipsis der Friesel dicitur,' Lips., 1655. (Résumé in Haller, 'Disput. med. pract.,' v, p. 449.)

among the group of symptoms; the most various forms of disease, in which papular or vesicular exanthemata occurred, came to be called "purples," until at last simple sudamina, which were often produced abundantly by the then fashionable diaphoretic practice, were brought within the compass of the term; and in the end there was such a confusion of ideas as to render all attempts at an understanding futile. This obscurity in the meaning of "*Friesel*" continued in Germany into the nineteenth century; and thus it happened that, when a disease that had been for the most part known only in France and Italy, under the name of "suette miliaire," came to be recognised about the beginning of the century in Germany, there was an opportunity for a further confusion of ideas, and the new disease was also received within the wide designation of "*Friesel.*" Prolonged inquiries on the part of French and German physicians have cleared up this question to the extent, at least, that the vague term of *Friesel* has been quite given up. The conviction has gained ground that the "Suette miliaire" of the French, for which I have proposed "Schweissfriesel" as a German equivalent, has nothing in common with all those forms of disease in the seventeenth and eighteenth centuries that were included under the designation of *Friesel*, and that in the *Suette miliaire* we have to deal with an infective disease, an acute fever mostly epidemic, which is characterised by the sudden outbreak of very profuse perspiration with a penetrating odour, by a feeling of severe constriction at the pit of the stomach, by want of breath, palpitations, gastric symptoms, splenic enlargements, sometimes even by cerebral symptoms, and by the breaking out, in the great majority of cases, if not in every case, of a rash, which is papular and vesicular, and occasionally bullous. This fever runs its course usually under eight days; it has a very mild character in most epidemics, but in some the deathrate has been 20 per cent. and upwards of the sick.[1]

[1] A detailed account of the pathology and treatment of miliary fever, with a retrospect of the whole literature of the disease, is given in an article by me in 'Virchow's Archiv,' viii, p. 454, and in my edition of Hecker's epidemiological writings ('Die grossen Volkskrankheiten des Mittelalters,' Berlin, 1865, pp. 363—392), to which I beg to refer the reader.

§ 22. History and Chronology of the Epidemics in France.

The *history of miliary fever* does not extend beyond the beginning of the eighteenth century.[1] The first unambiguous information about the disease dates from 1718, in which year, according to the statements of chroniclers, it was observed for the first time in various parts of Picardy, whence the name "*Suette des Picards.*" The malady soon showed itself in Normandy, and next it spread over certain districts of Poitou, Ile-de-France, Burgundy, and Flanders. As usually happens on the sudden outbreak of diseases previously unknown, the physicians of that period sought for the origin of the malady beyond the frontiers of their own country. Some laid the blame on pernicious winds blowing from the Netherlands coast over Northern France and carrying the pestilential poison with them (Bellot), while others explained the disease as having been introduced from abroad into the port of St. Valery (Ref. I). The repeated epidemic outbreaks of the *suette* during the years following, within the limits of the localities mentioned, soon showed that these assumptions were untenable, and they proved that it was really some local origin of the malady that had to be sought for. Up to the end of the century it remained limited to the North and East of France; at all events, according to the experience of Lorry, the South of France had escaped it altogether up to 1770. Miliary fever showed itself there first in 1772 and 1773, in Provence; it was still more widely spread in Languedoc in 1781 and 1782, and in the same years it was prevalent in the North-East of France to a greater extent than ever. Its area has gone on increasing in the course of the present century, so that the disease may now be looked upon as one of the most widely diffused of the endemic or epidemic diseases that France suffers from.

[1] The evidence adduced by Seitz and others (*see* the 'Bibliography') from the writings of the physicians of antiquity, as well as of those of the middle ages and the earlier centuries of the modern period, in order to prove that these writers were acquainted with miliary fever, is based, in my opinion, upon an erroneous conception of the morbid process, which is essentially characterised, not, as that author assumes, by the exanthem, the least constant of the phenomena, but by the profuse sweat and by the nervous symptoms before mentioned.

Chronological Table of the Epidemics of Miliary Fever in France from 1718 to 1879.[1]

Year	Department.	Place.	Season.	Authorities.
1718	Somme	Viemeu, Abbeville, Amiens, &c., in Picardy	Summer	
„	Aisne	St. Quentin	„	
„	Orne	At various places in Normandy	„	Ref. ii. Ref. iii.
„	Nord	In several communes of Flanders	„	
1723	Pas-de-Calais	Arras, &c., in Artois	„	Ref. iv.
„	Nord	Cambray and neighbourhood	„	Ref. v.
1726	Aisne	Melun	„	Vandermonde.
„	Seine-et-Marne	Guise	„	
1732	Seine-et-Marne	Meaux	Spring	Ref. vi.
1733	Somme	Abbeville and other places in Picardy	Summer	Bellot.
1734	Bas-Rhin	Strasburg	Aut. & Win.	Salzmann, Lindern (I).
1735	Seine	Near Paris	Sp. & Sum.	
„	Seine-et-Oise	Fréneuse, Vexin franç.	„	Quesnay.
„	Eure	Vexin normand	„	
1737	Orne	Argentan, Vire, Falaise, &c., in Normandy	—	Lepecq, p. 256, 323, 368, 419.
„	Calvados		—	
1738	Seine-Oise	Luzarche and Royaumont	Spring	Ref. viii.
1739	Aisne	Château-Thierry	—	Foucart, p. 305.
1740	Seine-Marne	Provins	Spring	Naudot.
„	Eure	Berthonville	—	Rayer, p. 446.
1741	Seine infér.	Rouen	Spring	Pinard.
1742	„	Caudebec	„	Lepecq. p. 156.
1747	Seine	Near Paris	Summer	Malouin.
„	Seine-Oise	Chambly & Beaumont	„	Vandermonde.
1748	Marne	Chalons s. M.	—	Navier.
1750	Aisne	Guise and Granvilliers	Summer	Ozanam.
„	Oise	Beauvais	—	Boyer.
1752	Seine-Oise	Etampes	Summer	Ref. ix.
„	Marne	Sermaise	„	Meyserey, p. 5.
1755	Allier	Cussett	Spring	Debrest (I).
1756	Pas-de-Calais	Boulogne s. M.	Summer	Desmars.
1757	Puy-de-Dôme	In the years 1757–62 in Low. Auvergne more or less extensively	„	de Pleigne, Brieude
1758	Nord	Lille	„	Boucher (I).
„	Calvados	Falaise	Spring	Lepecq, p. 156.
„	Allier	Vichy	Winter	Aufauvre.
1759	Seine infér.	Caudebec	Summer	Lepecq, p. 156.

[1] The references in the column "Authorities" are to writings quoted in alphabetical order in the list of authors at the end of the chapter.

Year	Department.	Place.	Season.	Authorities.
1759	Aisne	Guise and vicinity	Summer	Vandermonde.
„	Allier	Gannot, &c., in Cusset	Spring	Debrest (II).
„	Oise	Compiègne	—	Bida.
1760	Orne	Alençon	—	Lepecq.
1763	Calvados	In many localities	Summer	Lepecq, p. 347, Ref. x.
„	Seine-Oise	Etampes	Spring	Boncerf.
„	Nord	Lille	Summer	Boucher (II).
1764	Seine-Oise	Angerville, near Etampes	Spring	Boncerf.
1765	Calvados	Caen (especially Vorst. S. Sauveur)	—	Lepecq.
1766	Manche	Avranches	Summer	Lepecq.
„	Orne	Laigle	„	Lepecq.
1767	„	Tinchebray	„	Lepecq.
„	Calvados	Caen and vicinity	Spring	Lepecq.
1768	Manche	Avranches	Autumn	Lepecq.
1769	Allier	Chambon de Combrailles and vicinity	Spring	Barailon.
1770	„	Chambon de Combrailles and vicinity	Winter	Barailon.
„	Calvados	On the Coast	Summer	Lepecq.
„	Eure	Lonviers	„	Lepecq.
„	Manche	Avranches	Autumn	Lepecq.
1771	Loiret	Montargis	Summer	Gastellier.
1772	Basses Alpes	Forcalquier	—	Bouteille.
1773	„	Ornison, &c., in Provence	—	Bouteille.
„	Seine infér.	At various places	Summer	Lepecq, p. 109, 135.
„	Nord	Lille	Spring	Boucher (III).
„	Oise	Beauvais and vicinity	Winter	Tessier.
1774	Calvados	Harcourt	Spring	Lepecq, 139.
„	Allier	Chambon de Combrailles	Winter	Baraillon.
1775	Manche	Avranches, &c.	Summer	Lepecq.
1780	Seine-Oise	Corbeil	Winter	Rayer, p. 435.
1782	Aude	Castelnaudary, Castres, St. Papoul, Carcassone, Toulouse, Laveur, Perpignan, &c., in Languedoe	Sp. & Su.	Pujol, Duplessis, Ref. xi.
„	Tarn			
„	Haute-Garonne			
1783	Oise	St. Reinan (Beauvais)	—	Rayer, p. 435.
„	Saine-Oise	Falaise, Beaumont, &c.	Summer	Ibid.
„	Seine-Marne	In many villages	Spring	Ibid.
1784	Rhône	St. Foix, near Lyons	—	Reydellet.
1791	Oise	Méru, Corbeil, &c.	Winter	Poissonier.
„	Nord	Douay	Autumn	Taranget.
1810	Oise	Beauvais and vicinity	Sum. & Aut.	Rayer, p. 437.
1812	Bas-Rhin	Rosheim and vicinity	Spring	Schahl & Hessert, Schweighäuser.
1817	Seine infér.	Arrond. of Yvetot	—	Lefèbure.
1820	Bas-Rhin	Dorlisheim	Summer	Foderé, p. 78.
1821	Oise	At many places over a wide area	Sp. & Su.	Rayer, Moreau, Francois, Dubun (I).
„	Seine-Oise		„	
1822	Somme	Near St. Valery	Summer	Ravin.

Year	Department.	Place.	Season.	Authorities.
1830	Seine infér.....	Arrond. of Yvetot ...	—	Lefèbure.
1831	Vosges	Plombières	Winter	Turck.
1832	Oise.....	General diffusion.....	Spring	Menière, Hourmann.
,,	Haute-Marne..	Chaumont	Summer	Robert (I).
,,	Pas-de-Calais..	Auxi-le-Chaumont ...	,,	Defrance.
,,	Seine-Oise	Several villages	Sp. & Sum.	Bazin, Delisle, Dubun (II).
,,	Dordogne	A few communes.....	—	Parrot (I).
,,	Haute-Saône...	Vesoul	—	Pratbernon.
1833	Bas-Rhin	Rosheim.....	Winter	Maugin.
1835	Dordogne	Canton Mareuil	—	Parrot.
1837	Haute-Saône...	Vesoul	Spring	Pratbernon.
1838	Vosges	Plombières	Summer	Turck.
,,	Aisne	A few communes of the Arrond. Laon...	—	Lejeune.
1839	,, ...	Bellicour	—	Bourbier.
,,	Seine-Marne ...	General diffusion.....	Spring	Barthez, Bourgeois (I).
1841	Dordogne	General diffusion.....	Sp. & Sum.	Parrot, Borchard, Galy, Pindray, Pigné, Rayer (III), Martin-Solon.
,,	Charente	General diffusion.....	,,	Gigon, Genueil, Rayer, Martin-Solon, ll. cc.
,,	Gironde	Bordeaux	Summer	Mignot, Chabrely, Ref. xii.
,,	Manche	Coutances	,,	
1842	Lot-et-Garonne	At very many places	,,	Burtez, Martin-Solon.
,,	Tarn-Garonne.	In a few villages	,,	
,,	Jura	In a few villages	Su. & Aut.	
,,	Deux-Sèvres ...	In a few villages	Winter	
,,	Haute-Saône...	In a few villages	Summer	
,,	Eure	Bernay	Spring	Ref. xiii.
1843	Marne.....	La Fère champenoise	,,	Martin-Solon, l. c.
,,	Bas-Rhin	Geipolsheim	Winter	Reibel.
1844	Vosges	Nothalten	—	Tauflieb.
,,	Oise.....	.....	—	Verneuil.
,,	Somme	Abbeville	Spring	Martin-Solon, l. c.
1845	Haute-Marne..	To a small extent.....	Summer	Martin-Solon, l. c.
,,	Vienne	Arrond. of Poitiers ...	,,	Id., Arlin, Loreau, Gaillard, Morineau.
1846	Cantal.....	The village of Chaudesaignes	,,	
,,	Hérault	Arrond. of Bezières...	Su. & Aut.	Martin-Solon, l. c.
,,	Doubs.....	To a small extent.....	Winter	
,,	Var	Arrond. of Brignoles	—	Ref. xiv.
1847	Haute-Saône...	Breurey (Arrond. Vesoul)	Wint. & Sp.	Sallot.
1849	Somme	In general diffusion...	Spring	Foucart, Bucquoy, Guérin.
,,	Seine-Oise	Noyon, Etampes, &c.	Sp. & Sum.	Bourgeois (II), Colson.

Year	Department.	Place.	Season.	Authorities.
1849	Oise.....	Compiègne, Chambly, &c.	Sp. & Sum.	Foucart, Vernueil, Tourrette, Gaultier.
„	Aisne	In general diffusion...	Spring	Foucart.
„	Marne.....	Sézanne, Epernay, Fontenay, &c.	„	Reveillé - Parise, Boinet, Guérin.
„	Meuse.....	The vicinity of Verdun	Summer	Guérin.
„	Jura	A few communes of the Arrond. of Dôle	„	Gaultier.
„	Deux-Sèvres ...	Several places in the Canton Niort	Spring	Id.
„	Haute-Saône...	A village in the Canton Bray	Winter	Id.
„	Gers	Arrond. of Condom...	Spring	Jägerschmid, Gaultier.
	Yonne.....	Tonnerre	Summer	Lachaise, Badin et Sagot.
„	Bas-Rhin	Andlau, Nothalten, &c.	Winter	Tauflieb, Ref. xv.
„	Meurthe.....	Arrond. of Château-Salins	Summer	Simonin.
„	Moselle	Thionville	„	Allaire.
„	Puy-de-Dôme..	Several communes ...	„	Nivet et Aguilhon.
1850	Seine infér.....	Cailleville (Arrond. of Yvetot)	Winter	Lefebure.
1851	Manche	Arrond. of Valognes, &c.	Sp. & Sum.	Gaultier (II), p. clxix.
„	„	Carentan	Summer	Id., p. clxxi.
„	Somme	At several places in the Arrond Roisel	„	Id., p. clxxii, Bucquoy.
„	Lozère	Arrond. Florac at several places	Sp. & Sum.	Id., p. clxv.
„	Hérault	Arrond. of Pézenas...	„	Id., p. clxxiv, Grynfelt.
„	„	Arrond. of Béziers ...	—	Bernard.
1852	Eure	Two villages in the Arrond. Bernay ...	Summer	Gaultier (III).
„	Jura	One village in Cant. Montmerey	„	Id.
„	Lozère	Arrond. of Mende.....	—	Marie.
„	Bas-Rhin	Weyer.....	—	Stöber et Tourdes, p. 414.
1853	Jura	Lons-le-Saulnier	Winter	Gaultier (IV).
„	Bas-Rhin	Altenweiler	—	Stöber et Tourdes.
„	Haute-Marne	In many villages	—	Vergne.
„	Seine-Marne			
1854	Bas-Rhin	Düttlenheim.....	—	Stöber et Tourdes.
„	Oise.....	In considerable diffusion.....	—	Barth (I), p. exxvi.
„	Marne.....	Etréchy	Summer	Chalette.
„	Vosges	Arrond. Neufchâteau	„	Destrem, Jacquot.
„	Haute-Marne..	At many places	„	Barth (I), p. clxv, Jacquot, Foucart (II).
„	Haute-Saône...	Arrond. of Pesmes ...	„	Bertrand.

Year	Department.	Place.	Season.	Authorities.
1854	Jura	Arrond. of Dôle	—	Chauvin.
,,	Isère	At a Monastery in Viriville	Summer	Barth (I), p. clxii.
,,	Haute-Garonne	In general diffusion...	,,	Foucart (II).
,,	Lozère.....	Arrond. of Marvejols	Spring	Barth (I), p. clxiv.
,,	Aube	In general diffusion...	Summer	Dechambre, Hullin
,,	Côte-d'Or	At many places	,,	Dechambre, Clausse.
1855	Marne.....	Arrond. of Chalons and Epernay.....	,,	Barth (II), p. clxiv.
,,	Loire	A village in Montbrison	,,	Id., p. clviii.
,,	Meurthe.....	Arrond. of Château-Salins and Luneville	,,	Id., p. clxv.
,,	Bas-Rhin	Mutzig and Canton Molsheim	—	Stöber et Tourdes, p. 414.
,,	Jura	Longwy and Chaussin	—	Barth (II), p. cliv.
,,	Hérault	Arrond. of Béziers and Montpellier	Sum. & Aut.	Id., p. cxlvii.
,,	Charente	Three villages in the Arrond. of Cognac	Autumn	Id., p. cxvi.
,,	Landes	In many villages	—	Id., p. clvi.
,,	Haut.-Pyrénées	Arrond. of Bagnères	Autumn	Id., p. cxcii.
,,	Bass.-Pyrénées	At many points	Sum. & Aut.	Id., p. cxciii, Rossoutrot.
1856	Bas-Rhin	Neuhof	Winter	Robert (II).
,,	Lozère	In a few villages of the Arrond. of Marvejols	—	Ref. xvi.
1857	Indre-Loire ...	Arrond. of Tours	Spring	Haime, Meusnier.
,,	Nièvre	Arrond. of Château-Chinon	—	Duboz.
,,	Saône-Loire ...	Arrond. of Lonchans	—	Guillemont.
1859	Dordogne	Arrond. of Périgneux	Spring	Jolly (I).
1860	Var.....	Draguigan & vicinity	,,	Ref. xvii—Dumas, Boyer-Goubert.
1861	Dordogne	Nontron.....	—	Jolly (II).
1864	Aude	Arrond. of Castelnaudary	Summer	Galtier.
1865	Hérault	St. Chinian	Autumn	Coural.
1866	,, ...	St. Chinian	Spring	Coural, Briquet.
,,	Aisne	.....	Summer	Plouviez.
,,	Pas-de-Calais			
,,	Seine-et-Marne			
,,	Lot	In 34 villages	,,	Briquet.
,,	Doubs.....			
,,	Landes			
,,	Puy-de-Dôme..	In one village	Spring	Teilhol.
,,	Indre-et-Loire.	In one village	Summer	Meusnier.
1870	Haute-Garonne	In several villages ...	Spring	Nelé.
1871	Indre-et-Loire.	Arrond. of Tours	Winter	Meusnier.
,,	Haute-Garonne	In one village	Summer	Gailhard.
1874	Puy-de-Dôme..	Arrond. of Clermont	Spring	Mazuel.

According to the above table, 194 epidemics of miliary fever have been recognised on French soil within the period from 1718 to 1874;[1] of these the larger number have been limited to a single village or to a few localities, while others have been prevalent over wider areas, spreading even over whole districts or departments, and by their coincidence in time, particularly in the years 1832, 1842, 1849, 1853, 1854, and 1855, they have imparted a pandemic character to the disease. The incidence of these 194 epidemics proves, however, that some parts of the country have been peculiarly subject to the malady, in contrast to others which have remained free. Thus we find that out of the eighty-nine departments (not counting Nice, Savoy, and Corsica), there have been fifty-five affected by the disease hitherto; in twenty-one of these there has been only a single epidemic, in six there have been only two epidemics, in six others the disease has been a good deal more prevalent (Haute-Marne, Lozère, Pas-de-Calais, Puy de Dôme, Indre et Loire, and Haute-Garonne), while it has broken out comparatively often in fifteen (Aisne, Seine infér., Calvados, Manche, Somme, Nord, Marne, Vosges, Jura, Haute-Saône, Allier, Seine-Marne, Eure, Orne, Dordogne, Hérault), and in three it has been positively endemic (Seine-Oise, Bas-Rhin, and Oise). If now we place together those districts that have been attacked with especial frequency, we shall find that the disease has been very nearly confined to a strip of territory in the north-east of the country, extending from Franche-Comté through Alsace, Lorraine, the northern parts of Champagne, Flanders, Picardy, Ile-de-France, and Normandy, and including more particularly the departments of Jura, Haute-Saône, Bas-Rhin, Vosges, Haute-Marne, Marne, Nord, Pas-de-Calais, Somme, Aisne, Oise, Seine-Marne, Seine-Oise, Seine, Eure, Seine infér., Orne, Calvados, and Manche. One hundred and twenty-six out of the one hundred and ninety-four epidemics have occurred within the limits of that region, while the remaining sixty-eight have broken out (mostly at isolated spots, although sometimes they have become widely spread) in a few regions of middle

[1] The epidemics of miliary fever in Alsace and Lorraine having occurred at a time when these provinces were still French, I have counted them among the epidemics observed in France.

and southern France, particularly in Auvergne, in the adjoining department of Allier, in the Dordogne, and in Poitou; so that in the north-eastern group, including the three departments specially emphasised, the disease may be said to have had an endemic character.

§ 23. Italian Epidemics.

The first accounts of *miliary fever in Italy*, according to the writings of Fantoni and Allioni, date almost from the same period when attention was first called to the disease in France. The earliest records, relating to the disease in 1715-1753 in various parts of Piedmont, have not the trustworthiness that could be wished, as there has plainly been a confounding of exanthematic typhus, scarlet fever, and other diseases with miliary fever. The accounts of an epidemic in 1755 at Novara (*Allioni, de Augustinis*) may be taken as referring to miliary fever with more certainty, and still more so the statements of Damilano as to the wider extension of the disease in Piedmont in 1774. Among later accounts of the miliary fever in that part of Italy, there is one for the year 1817, when it broke out in Novara at the end of summer after the extinction of an epidemic of typhus (*Ramati*), and for the years 1821-23, when it attacked several villages in the province of Alessandria, in the district situated between Sale and Camerane (*Dalmazzone*). When and to what extent miliary fever spread from the Sardinian territory over other parts of Upper Italy cannot be decided with certainty, from the scanty and in part hardly trustworthy records; it would appear that the disease first became generally prevalent there towards the end of the second decade of the nineteenth century. Beyond the Sardinian Kingdom we meet with the malady first at Corregio (Modena), where it was epidemic in the summer of 1775 (*Baraldi*); next in Venetia, where it broke out first in 1790 at Verona, and is said to have spread thence westwards (*Pollini, Arvedi*); it was epidemic at Vicenza in 1817, somewhat later at Treviso, towards the end of the fourth decade at Padua,[1] still later at Venice (*Taussig*), while in

[1] Lippich ('Advers. Med.-Clin.,' Ser. ii, Fasc. i), speaks of miliary fever as disease unknown at Padua in 1835.

several districts of Friaul it had been observed to be epidemic as early as the spring of 1835 (*Podrecca*). In the plain of Lombardy the disease appeared first at the beginning of the century on the banks of the Po (*Jemina*), especially in the district of Mantua, afterwards in the adjoining district of Brescia (*Menis* I, p. 152), and not until the fifth decade did it penetrate to Milanese territory, to Pavia (*Pignacca*), and Cremona (*Tassani*). Thus, a small epidemic of miliary fever was observed by Storti in the summer of 1844 in the village of Pomponusio; it was prevalent at Pavia in the hot summer of 1846, and in the district of Borgosatollo (Mantua) it was observed from the spring to the autumn of 1848 (*Belpietro*). In 1854 it broke out as an epidemic at several places in the province of Brescia, and it reappeared there in the summer of 1856, being especially malignant in Carpenedolo (*Maraglio*). The most recent accounts of miliary fever come from Tuscany. In that province the disease showed itself first at Florence in the winter of 1836-37, according to Seitz, who follows Zink; in 1843 and 1844 it broke out there anew, and two years after it spread thence to Pisa, Leghorn, Poggibonzi, Fauglia, and other places (*Taussig*), since which time it has been repeatedly epidemic in those parts, particularly in 1853 at Sangimignano (*Cantieri*), in 1854 at Ponte a Cappiano (*Tempesti*), in the winter of 1855-56 at Poggibonzi (*Burresi*) and Fauglia (*Gattai*), and in 1858 and 1859 again at Sangimignano (*Cantieri*). In the summer of 1861 it was prevalent at Dovadola, imported, as Liverani explains, from Terra-del-Sole, after having been epidemic at Forlì for a year previously; in the autumn of 1872 the sickness was observed, according to the account of Borgi, in Galleno (circondario of San Miniato), and in the summer of 1875 at Isernia. Fazio, to whom we owe the account of the last-mentioned epidemic, states that there could be no doubt of the epidemic prevalence of the disease in the Aemilia, in some districts of Lombardy and Piedmont, and particularly in Tuscany. Whether Central and Lower Italy have hitherto escaped the miliary fever altogether, I have not been able to decide. The account by Palmieri[1] of a presumed epidemic of miliary fever

[1] 'Relazione e semplice cura della febre migliare, che ha regnata in Bevagna, &c.' Fuligno, 1805.

in 1804 at Bevagna (delegation of Perugia) is not known to me; according to the summary of it by Corradi,[1] that epidemic was in nowise concerned with the disease of which we are now speaking.

§ 24. Other Epidemics, mostly German.

Besides France and Italy, the only other country in which miliary fever has attained the importance of an epidemic disease is *Germany*, and particularly its south-western divisions. Disregarding the altogether ambiguous accounts of "Friesel" on German soil during the 18th century,—accounts from which, it is impossible, as we have seen, to obtain at the present day any definite notion of what disease they had before them—we first meet with the disease in a few minor epidemics, as at Wittenberg in the winter of 1801 (*Glüser, Kreyssig*), next in the spring of 1820 at a village in the neighbourhood of Bamberg (*Speyer*), and at the small town of Giegen in the Würtemberg district of Heidenheim (*Steudel*, p. 92), and in the summer of 1825 in a few parishes of the Bavarian justiciary district of Erding (*Seitz*, p. 334). The first accounts of a wider diffusion of miliary fever in Germany date from the period between 1828 and 1836, or from the time of the more general prevalence of the *Suette miliaire* in France, and the first outbreak of cholera in Europe; and during that period, as well as in previous years and subsequently, it was in the southern part of the country, more particularly in the south-west, that the disease was found to be especially common or to have the character of an endemic. In Würtemberg, miliary fever showed itself in the spring of 1829 at Oeffingen (*Steudel*, p. 77), and at Ensingen (*Schnurrer*), in the spring of 1830 in and around Mettingen (*Steudel, Manz*), in the winter of that year and in the spring of 1833 at several villages in the district of Gmünd (*Bodenmüller* I), and in the winter of 1832-33 in the district of Vaihingen (*Keyler*). In Baden it broke out as an epidemic in 1828, 1833, 1835, and 1836 at several places in the circles of Main and Tauber (Ref. XIX), and in 1839 at Renchen

[1] 'Annali delle epidemie occorse in Italia, &c.,' Parte iv. Bologna, 1877, p. 568.

(*Schaible*). In Bavaria it was prevalent during the spring and summer of 1828 in the Roththal (on the western border of the circle of the Upper Danube, *Beck*), in the winter of 1833 in several villages of the justiciary district of Weilheim (*Seitz*, p. 358), and in May, 1834, at a village in the neighbourhood of Würzburg (*Fuchs*). In the years following, the disease was again not uncommon in the above localities, but it was mostly found in more isolated epidemics, often limited to quite small areas; thus, in February, 1837, it occurred in two parishes of the Würtemberg district of Gmünd (*Bodenmüller* II), in the spring of 1838 at Herlheim in Lower Franconia (*Stahl*), and in 1842 at Passau (*Egger*); but it was especially prevalent in the summer and autumn of 1844, when it spread from the justiciary province of Neumarkt, which had been often visited by it, to the districts of Mühldorf, Altötting, Vilsbiburg, Erding, Landshut, Dingolfing, and Landau, over a hilly tract of country sixteen leagues long and fourteen broad, situated partly in Lower and partly in Upper Bavaria (*Seitz* II, *Ebersberger*, *Egger*). Besides Würtemberg and Bavaria, it is chiefly in some of the mountainous parts of *Austria* that the disease has been found as an epidemic from time to time during the last thirty or forty years; thus, in Styria in the summer of 1835 (*Vest*), and afterwards, in the sixties, in the Lasnizthal, where accounts make the disease to be quite endemic in several of the hilly parts (*Macher*); further, in Upper Austria in the summer of 1836, in the spring of 1839 at Tarnow in Galizia (*Kellermann*), and in a few of the mountain hamlets of the Saaz circle in Bohemia (*Müller*), and more recently, in the autumn of 1859, in the small town of Ybbs in the circle of the Wiener Wald (*Masarei*). In the epidemiological records of Central and Northern Germany, mention is indeed often made of "Friesel" and epidemics of the same, but besides the small epidemic at Wittenberg in 1801 already referred to, only a few of them can, on the evidence, be assigned a place in the history of miliary fever. Such were the outbreaks in a village near the town of Meiningen in the summer of 1833 (*Jahn*), in the Kalau circle, province of Brandenburg, in the autumn of 1838 (*Roedenbak*), in the small town of Frauenstein in the Saxon Erzgebirge in

the winter of 1839,[1] and finally, in the winter of 1849 in the village of Wegeleben, circle of Oschersleben (*Andreae*). To these few data may be reduced all that is known of the occurrence of the disease in Germany.

The accounts of miliary fever in *Switzerland* which have come down from the middle of last century are highly uncertain,[2] and equally unauthentic are the statements of French newspapers as to the occurrence of the disease in 1849 in Biscaya, *Spain*. So that, in addition to France, Italy, and Southern Germany, we find on the soil of Europe, as well as on the globe generally in so far as its conditions of disease are known to us, only one point where miliary fever has attained a degree of importance, and that but a slight one, viz. *Belgium*. The disease appeared there first in 1838 in a few communes of the Hennegau,[3] next in 1849, along with cholera, at Liege, Namur, and in the vicinity of Mons (*Leynseele*), the year after at Hotton in Luxembourg (*l'Hermitte*), and finally in 1866, again in conjunction with cholera, in epidemic diffusion throughout many parts of Luxembourg (Ref. XVIII).

§ 25. Limited Area.

Few of the acute infective diseases have been so narrowly circumscribed in their *geographical distribution* as miliary fever. Even within those narrow limits the disease has been mostly epidemic at scattered points only, or confined to a single village or a group of villages, seldom spreading over wider circles or over large tracts of country. This behaviour of miliary fever comes out most clearly in Germany and Belgium; in Italy also, if one may judge from the very incomplete data at our service, the disease appears to have been commonly prevalent in isolated or somewhat restricted epidemics. In France alone has it overrun wide districts in certain years; as in 1757-62 in Lower Auvergne, 1772-73 in Provence, 1821 in the departments of the Oise and Seine-et-

[1] 'Physikatsbericht im Königreich Sachsen,' 1839, p. 69; 1840-41, p. 163.

[2] See especially Allioni (German transl.), p. 19.

[3] Meynne, 'Topogr. méd. de la Belgique,' 1865, p. 234.

Oise, 1832 in the Pas-de-Calais, Seine-et-Oise, Oise, Haute-Marne, Haute-Saône, and Dordogne; 1841-42 in the Dordogne, Charente, Gironde, Tarn-et-Garonne, Jura, Deux-Sèvres, and Haute-Saône; but particularly in 1849, 1853-55, and 1866, when the spread of miliary fever over a great part of France gave the disease almost a pandemic stamp.

§ 26. A Disease of Short Duration.

This character of limited diffusion in most of the miliary fever epidemics, has its counterpart in their strikingly *short duration*, wherein they correspond exactly to the English sweating sickness, and strongly remind us of influenza and dengue. The mean duration of epidemics of miliary fever at a given place has amounted to three, or at the most four weeks, and not unfrequently it has not exceeded a space of seven or fourteen days.[1] Even in those cases where the sickness was protracted over two or even three months, it usually happened that, during the first weeks, only a case showed itself here and there, then all at once a large number of persons would sicken, the epidemic quickly reaching its height and as quickly subsiding; so that the duration of the disease in its proper epidemic prevalence was in fact limited to some two, three, or four weeks, while there was a further considerable period in which single cases occurred here and there until the outbreak came to an end.[2]

[1] The epidemic of 1833 in the neighbourhood of Meiningen lasted eight days, that of 1843 at Geipolsheim ten days, and those of 1801 at Wittenberg and 1851 at Busson (Hérault) fourteen days.

[2] Thus, we read in the account by Pujol (l. c.), p. 274: "There is another peculiarity presented by our epidemic, which does not appear to have been observed elsewhere in so marked a manner. In the towns to which the miliary epidemic travelled, one met at first with a few individuals here and there who had the disease sporadically. But the moment the sickness became truly epidemic, people began all at once to be laid up with it by the hundreds every day, the number attacked within the twenty-four hours being greater every day for the first few days. After six or seven days the daily number of subjects attacked began to diminish, and went on decreasing for six or seven days longer, so that at the end of fifteen or sixteen days the miliary fever ceased to be epidemic and resumed its sporadic course as at first." Precisely the same sequence of events marked the epidemic of 1832 in the department of the Seine-et-Oise, and also that of 1859

Among those epidemics wherein the disease had attained a somewhat wider diffusion over large areas, there have been many differences noticed in its manner of beginning and of spreading, as well as in the length of time that it has continued to spread. It would not unfrequently happen that many villages within a certain radius would be attacked by the disease all at once; another time the sickness would spread to various sides along the radii, and as if from a centre, but without attaining an equal speed in all these several directions, or travelling equally far; in still other cases, the diffusion has taken place by leaps and bounds; or finally, the epidemic has travelled quickly in a particular direction, then remained stationary for a time, and after a considerable interval again travelled forwards, or it might be backwards. Thus, to take the example of the epidemic of 1841 in the Dordogne, the disease appeared first in June in the arrondissement of Nontron, in the north-west of the department, and from there it spread in a south-easterly direction to Marcuil and as far as the Drône, appearing the same month in a few communes on the left bank of the stream, although in very mild form; but in July the miliary fever broke out in somewhat malignant form in localities that had hitherto remained exempt, and it now travelled very slowly in a southerly direction, so that at Périgueux, the most southern of the places invaded by the epidemic, the first cases occurred at the beginning of September, the height of the epidemic fell in the middle of that month, and the end of it in the first days of October.

A very striking contrast to this narrow limitation which miliary fever has usually been subject to in its epidemic prevalence in time and place, is presented to us in the *remarkable extent that the epidemic frequently attains in the places which it visits*, an extent in some cases so considerable that influenza alone of common epidemic disease affords an analogy. Thus Pujol states that in the epidemic of miliary fever in 1782 throughout part of Languedoc, the number of the sick amounted to 30,000; in the epidemic of 1772 at

at Ybbs; the latter lasted from the 15th September to the 31st of December, but, as Masarei remarks, by far the larger number of cases occurred in the period from the 1st to the 15th October.

Forcalquier (Provence), with a population of 2000, there were 1400 cases, or 75 per cent.; and at Busson (Hérault) in 1851, almost the whole population (or 800 out of 1000) were attacked by the disease in a space of two weeks. A rate of sickness of 25 to 30 per cent. of the whole population has often been observed; the average numbers attacked in miliary fever epidemics, so far as we can judge from the data before us, may be put at 10 to 20 per cent. of the population; while at the same time there have been not unfrequently epidemics with a sick rate of not more than 2 to 9 per cent.

Not less great than these variations in the ratio of sickness in epidemics of miliary fever, have been the fluctuations in the mortality of the disease at various times and in various places. Miliary fever in general is to be accounted a disease seldom fatal, and not a few epidemics can be adduced in which the mortality was zero; such were the outbreaks in 1821 in the Seine-et-Oise, 1849 in the Oise, 1851 at Busson (Hérault), 1853 at Boulogne (Haute-Marne), 1854 in the Vosges and Haute-Marne, and 1855 at Cognac (Charente). In others the mortality has amounted to not more than 1 to 5 per cent. of the sick; as in 1821 in the Oise, 1842 in the Dordogne and most parts of the Lot-et-Garonne, 1851 in Carentan, and in 1855 in the arrondissement of Bagnères. On the other hand, we know of not a few epidemics in which the mortality reached the figure of 6 to 13 per cent., notably in 1812 in the Bas-Rhin, 1832-33 in the Seine-et-Oise, 1839 in the Seine-et-Marne, 1841 in the Dordogne, 1849 at Niort, Dôle, &c., 1851 at Florac, 1860 at Draguigan, 1854 at Ponte a Capiano, 1801 at Wittenberg, and 1844 in Bavaria. Finally, there are a number of statistical facts showing a mortality of 15 to 30 and even up to 50 per cent. of those attacked by epidemic miliary fever; but that proportion is obviously exceptional, and it has been found most frequently where the epidemic has been very limited in extent.

§ 27. Close Dependence on Season and Weather.

Among the factors which exert an appreciable influence upon the production of miliary fever, the foremost place is unmistakeably taken by the *season of the year* and the *weather*.

Among the 184 epidemics in the synopsis given above, in which the time of appearance and prevalence of the disease is accurately stated, the seasons were as follows:

46 in spring.	6 in summer and autumn.
16 in spring and summer.	8 in autumn.
1 in spring, summer, and autumn.	1 in autumn and winter.
77 in summer.	27 in winter.
	2 in winter and spring.

Of the 184 epidemics, there were accordingly 83 beginning in summer and 63 in spring, while only 29 began in winter and 9 in autumn, so that nearly seven-ninths of all the epidemics ran their course during spring and summer. The reason for the prevalence of miliary fever in those seasons will have to be sought for in the conditions of weather proper to them, and in fact it appears that a high temperature liable to sudden changes and associated with a large amount of moisture in the air is especially favorable to the outbreak and prevalence of the disease. It is a noteworthy fact in this connexion that most of the winter epidemics of short duration (two to three weeks), such as those of Vichy in 1758, Douay in 1791, Wittenberg in 1801, Esslingen in 1831, Rosheim in 1832, and Weilheim in 1833, broke out and ran their course at a time of very relaxing and damp weather.

§ 28. Influence of Locality.

The influence of *locality*, particularly of the *soil*, on the occurrence and diffusion of miliary fever is not so obvious. Many observers lay much stress upon the prevalence of the disease on damp or marshy ground. At the first outbreak of the disease in 1718, in Picardy, it was pointed out that the epidemic spread along a damp valley on a peat soil, while the adjoining plain, with a dry chalk bottom was

unaffected; it was remarked, further, that miliary fever often occurred at Cusset, situated in a damp valley; and it was shown that the epidemic of 1782 in Languedoc travelled exclusively along the Canal du Midi, and that the sickness of 1772 and 1773 in Provence, as well as that of 1812 in Alsace, was confined to deep and damp valleys, in the one case of the Alps and in the other of the Vosges, the high and airy localities having been affected very little or not at all. When the disease was prevalent in the vicinity of Bamberg in 1820, attention was directed to the low and damp situation of the locality attacked; in the Roththal in 1828 the epidemic was restricted to the low-lying and marshy villages, while the high ground escaped. The outbreak of miliary fever in 1829 at Ensingen was preceded by an inundation of the marshy region. The situation of Herlheim, where the disease broke out in 1838, is a wet moor. Barthez found in the epidemic of 1839, in the canton of Rebais, that the villages situated in a close valley much subject to inundations were principally affected. It was proved that the sickness of 1841 in the Charente was particularly prevalent on the marshy banks of the Lione, and that it decreased in the extent of its diffusion and in its severity the farther inland it travelled. When it was prevalent in Bavaria in 1844, it spared the lofty and dry situations and confined itself principally to the long valleys, damp, marshy, or boggy, and enclosed by a circle of hills. In like manner, in the epidemics of 1844 and 1849 in the department of the Oise, it was principally the damp and marshy places that were visited; and the same etiological factor was conspicuous in the 1849 epidemic at Thionville and around Vesoul, and again in 1851 at Carentan, in 1856 at Neuhof, in 1860 in Belgium, where the places affected by the inundations of the Ourthe were attacked first, and in 1859 at Ybbs where it was again the lower parts of the town, damp and exposed to frequent inundations, that suffered, along with some adjacent villages situated in wet meadows. In the department of the Puy-de-Dôme the villages to suffer most were those occupying the water-logged banks of the Limange, and in fact the outbreak of the disease was usually preceded, especially in the epidemic of 1866 at Davayat and in that of

1874 at Clermont, by considerable floods. However deserving of attention these facts must always be in estimating the pathogenesis, it should not be forgotten, on the other hand, that miliary fever has not rarely broken out under entirely opposite conditions of soil,—that it has avoided the low-lying, damp, or marshy localities to become prevalent on dry ground and on airy plateaus. Thus we find the disease dominant on the chalk soil of Northern France, dry on the whole and in part sterile; and on the rocky precipitous coast of Normandy (particularly Calvados). In the department of the Oise it was principally the elevated and dry localities that suffered in the epidemics of 1810, 1821, and 1832, in contrast to those of 1844 and 1849; and we find the same to be true of the disease as it occurred in 1820 at Giengen, and in 1830 at Mittingen and Gmünd, in 1841 in the Dordogne, 1842 in the Lot-et-Garonne, 1844 in Poitiers, 1849 in the departments of the Somme, Aisne, and elsewhere, 1851 at Peronne, 1853 at Menetaux, 1854 at Viriville, and in 1866 at Pernes-en-Artois in the Pas-de-Calais.[1] Finally, it has to be mentioned that in Upper Italy the disease showed itself first in the mountainous parts of Piedmont, and remained there for some time before there was any general diffusion of it over the plain of the country, while even in the latter situation it was by no means associated with a swampy condition of the ground.

§ 29. A Country Disease, but not particularly of the Peasantry.

The outbreak and diffusion of miliary fever may be shown to be entirely independent of the *neglect of hygiene* which goes alongwith the hardships of living. It is especially noteworthy in this connexion that the disease has appeared almost exclusively in small country communes, in market villages and such-like localities, while in larger towns it has been extremely rare and of brief duration, never attaining the importance of an

[1] In the account by Plouviez, it is stated (p. 36): "The epidemic broke out in a low and marshy locality overgrown with trees. Of the six communes attacked three had those conditions, while the other three, again, were much elevated above their neighbours, with little wood on them, dry and airy. Apart from considerations other than those of locality, the gravity of the sickness was always the same."

endemic disease, as it does in the former class of localities. The disease has been prevalent repeatedly in communes distinguished by their cleanliness, the adequate ventilation of their streets and houses, the well-being and sobriety of life of their inhabitants and by other favouring hygienic factors, whilst other communes near them, and far inferior to them in all hygienic matters, have escaped. Wherever it has appeared, it has attacked rich and poor in equal proportions; it has indeed happened not rarely that the proprietary class has furnished a larger contingent of the sick than those suffering from scarcity and want, as, for example, in the first Italian epidemics, in those of Strasburg in 1734, Novara in 1817, the department of the Oise in 1812, the Dordogne in 1842, Poitiers in 1844, the departments of the Somme, Seine-et-Oise, and others in 1849, and at Peronne in 1851. Aggregation of large masses of people in narrow and ill-ventilated spaces, such as barracks, prisons, hospitals, schools, and the like, so far from proving favorable to the development and diffusion of miliary fever, would even appear to have exerted an antagonistic influence. Thus Parrot[1] says of the epidemic of 1841 in the Dordogne: "Observation has shown in the clearest way that the more the individuals were crowded together, the smaller was the proportion of cases and the less serious their type. . . . . At Périgueux, all the establishments with a large number of inmates were spared: the barracks, in which two batallions were usually quartered, were without a single patient; the college, where the vacation did not begin until after the first eight days of the epidemic, had not a single pupil attacked; and in the prisons, which usually contained 100 to 120 persons, there were only three cases of the mildest kind." To the same effect Gaillard[2] speaks of the epidemic of 1844 in Poitiers: "Neither the inmates of the hospitals, or of the garrison, nor the applicants for public charity, were affected by it; well-to-do workmen, and persons belonging to the rich *bourgeoisie*, and to the tradesmen class, were the *sole* victims of the epidemic." In like manner, Bucquoy says of the epidemic of 1851 in the department of the Somme: "The miliary fever had this peculiarity, that it

[1] L. c., p. 191. [2] L. c., p. 51.

showed itself to be most severe wherever the hygienic conditions appeared to be the most favorable."

§ 30. A Specific Infection: Relations to other Diseases.

There is complete unanimity among observers as to the specific character of the process of miliary fever; but the views begin to diverge upon the question whether it is *a peculiar specific morbid poison* that we have to deal with, or whether the poison is to be regarded merely as a modification of the *marsh miasm*, that is to say, *malaria*. The supporters of the latter opinion seek to establish it by means of evidence that miliary fever is especially common, as we have already seen, on marshy and malarious soil, that a remittent type has been often observed in the course of the disease, and that the use of quinine in such cases has had a good effect. An impartial estimate of the facts already adduced in detail makes it at least highly probable that copious saturation of the ground brought about by heavy atmospheric precipitations or by inundations is an essential factor in the production of miliary fever, and that under these circumstances there may be processes set up within or upon the soil which stand in some relation to the development or cultivation of the morbid poison, the disease being in this respect allied to yellow fever, to cholera, to typhoid, and, indeed, to the malarial diseases themselves. But as we are in no wise justified in concluding that there is an identity of the morbid poison underlying all those diseases because they have one etiological factor in common, so there is just as little reason to conclude for the identity of malarial disease and miliary fever. With all the extensive diffusion which the latter has attained in France, it is precisely the great marshy and malarious regions of the country at the estuaries and in the lower basins of the Loire, Garonne, and Rhone, that have remained altogether exempt from or been rarely visited by it. In the epidemic of 1844 in Poitiers, the town itself situated on a dry limestone ridge, together with the dryest spots in its vicinity, suffered most, while the marshy districts in the neighbourhood were entirely exempt. The river valleys in the department of Hérault, in

which miliary fever has been often epidemic, are completely free from malaria, according to the testimony of Coural, Bernard, and others. In the epidemic of 1841 in the Dordogne, it was actually in the elevated regions with a chalk bottom that the disease was prevalent, while the adjoining swampy districts were unaffected; and although several chroniclers of the epidemic of 1849 in the department of Gers would make out the malarious soil of that tract of country to be the cause of the disease, still we cannot, in justice to facts, overlook the question why the marshes of Gascony, abounding in malaria, were for more than a century free from miliary fever, and on the whole so little visited by it when the disease did at length break out. The distribution of the disease also in Italy and Germany tells against the view here referred to. In the former, it is precisely the most intense foci of malaria on the swampy banks of the Po, especially the notorious marsh districts of Ferrara and Comacchio at the river mouth, that have been least visited by miliary fever. In Germany, again, the disease has been prevalent almost exclusively in those districts which must be counted among the least malarious in the whole country. The remittent type of the morbid process, which has been sometimes observed, will hardly justify the conclusion as to the malarious nature of the disease; still less the good effect of treating with quinine, which has, besides, been questioned by many.

Of the nature of the morbid poison itself, we are not able to form an opinion, and equally little are we able to determine the influences upon which depend modifications in the course of miliary fever epidemics, and above all, the amount of sickness and mortality in them. In many cases, the reason for the malignant character of the epidemic is to be looked for in an unsuitable therapeutic or dietetic practice, particularly in an irrational diaphoretic method; but in many other cases, this reason, often dwelt upon in too one-sided a manner, does not suffice to explain why the epidemic grows apace and why the sickness becomes of a more severe type. We are brought to a standstill here at those same confines of knowledge which mark the limit of our progress in the study of many other infective diseases—I mention only scarlet fever—and in

forming a conclusion on this matter we shall have to accept for the present the position of Bucquoy: "In the case of this as of most other epidemic maladies, everything is still mystery to us; after so many ages spent in research of every kind, we are still no farther forward than the *quid occultum, quid divinum,* of the father of medicine."

§ 31. Question of its Communicability.

The question of the *contagiousness* or *communicability* of miliary fever has been the occasion of lively controversies among French and Italian physicians. The reasons that have led Schahl and Hessert, Rayer, Loreau, Bucquoy, Robert, and others to answer this question in the affirmative are based chiefly upon the observation often made, that after one person has sickened of miliary fever, it is usual for the other members of the family to take the disease very soon, that in many cases the friends who have hastened to take care of the sick have become victims of the malady, and that in such cases the progress of the sickness from house to house can be followed, foci of disease being not unfrequently established from which the epidemic spreads or radiates. Foucart, who has taken the side of the contagionists, though not altogether definitely, thinks that an additional argument may be found for this opinion in the analogy, as he supposes, between miliary fever and the triad of acute exanthemata—smallpox, scarlet fever, and measles. The anti-contagionists, on the other hand, have pointed to the uniformly negative result of the experiments instituted by several physicians (Parrot, Borgi, and others), to inoculate with the contents of the miliary vesicles, which, however, proves nothing as to the non-contagious nature of the disease, inasmuch as the morbid poison may possibly reside in other secretions. They have further dwelt upon the comparatively rare occurrence of the disease in large towns, upon the more or less complete exemption from it, within the affected localities, of masses of people crowded together, the protection being in no wise due to isolation, and finally, upon the circumstance adduced by many observers in favour of

their view, that the children have for the most part remained healthy notwithstanding their close intercourse with sick members of the family, and that in innumerable cases the malady has not extended beyond *one* place, although that had been in free communication with the whole neighbourhood. If, then, we have regard to the mode of outbreak and course of the epidemic at particular places, as it has been described above, and to the manner of its diffusion on a large scale, there would seem to be the best reason for doubting the contagiousness or communicability ("transmission infectieuse," as Foucart cautiously expresses it) of miliary fever in general.

§ 32. Identity of Miliary Fever with the English Sweating Sickness.

A retrospect of the history of the English sweat and of the miliary fever, as here traced, leaves no doubt in my mind, of the close relations between the two diseases. Common to both are the sudden occurrence and the short duration of the epidemic, its prevalence in summer and autumn, the dependence of its origin upon the above-mentioned states of weather and corresponding states of the soil, the often observed immunity from it which the proletariat enjoys, the explosive outbreak of the disease, particularly in the night-time, the pronounced nervous symptoms as seen in the phenomena of anxiety, palpitation, want of breath (even to the extent of asphyxia and the like) the profuse sweats—the true *signum pathognomonicum*—the exanthem, which is for the rest as little constant in miliary fever as in the sweating sickness,[1] the frequent occurrence of relapses, the fatal termination ushered in by almost identical phenomena, and finally, the injurious effects of the diaphoretic treatment. If there is any doubt left of the close connexion between the two diseases, it may be dispelled by recounting the history of a

[1] I leave it undecided whether the occurrence of an exanthem in the sweating sickness was actually so rare as the scanty notices of it would lead one to suppose; whether, indeed, it had not rather escaped the attention of observers who had not looked for it, and escaped their notice all the more that an examination of the skin had been for the most part omitted for fear the patient should take cold.

slight epidemic of miliary fever in the autumn of 1802 at the small Bavarian town of Röttingen; not only the internal or pathological affinities of the two diseases, but also the exterior or epidemiological are brought out by it in so striking a way that we might reckon this Röttingen epidemic as belonging in an equal degree to the modern epidemics of miliary fever of a malignant type, and to the older form of the disease, the English sweating sickness.

The sickness broke out, according to the account given of it by Sinner,[1] in the end of November, after a long tract of rainy weather following upon a hot and dry summer; it lasted not quite two whole weeks, and was strictly confined to the town, all the country round having been entirely exempt. In this outbreak, as in others, it was chiefly strong people in the flower of their age that were attacked and prostrated, while the aged and the poorer class of people enjoyed a high degree of immunity.

The onset of the disease was sudden, with palpitation, pain in the back of the neck, and profuse sweats; in cases that ran an unfavorable course, there occurred "convulsive trembling of the whole body, swooning, and torpor," whereupon death followed, mostly within the first twenty-four hours. Besides the palpitation accompanied by a feeling of anxiety, want of breath was always observed. In the less severe cases, the nervous phenomena went off, and only the sweating continued, sometimes with the addition of exanthemata of various kinds ("blebs, spots, or miliary papules"), while the secretion of urine was mostly suppressed. Such cases would usually end fatally, if, as not unfrequently happened, there was a relapse of the severe symptoms already described. When the issue was towards recovery, the perspirations diminished gradually up to the sixth day, after which the patient entered upon convalescence in a state of great weakness.

The mortality at the beginning of the epidemic was appalling; and, as in other instances, it was materially assisted by the mistaken diaphoretic treatment: it is in fact an open question whether that was not the sole cause of the excessive death-rate. The observer to whom we owe the

[1] 'Darstellung eines rheumatischen Schweissfiebers, welches zu ende November, 1802, in dem churfürstlich-würzburgischen Städtchen Röttingen a. d. Tauber endemisch herrschte,' Würzburg, 1803. An exact reprint of this short but interesting tract will be found in the collection of Hecker's epidemiological works edited by me (Berlin, 1865, pp. 338—348).

account of it, arrived only a few days before the sickness came to an end; but, along with a practitioner who had already hastened to give his help—in the town itself there had been no medical attendance procurable up to that time—he instituted a suitable treatment, more cooling but still within such limits as to obviate a chill, with a strengthening diet, and medicines for the period of convalescence; and from that time only one case of death was observed.

§ 33. Coincidence with Cholera Epidemics—"Cholera Sudoral."

The history of epidemic sweating diseases is of special interest from one other point of view: *the coincidence in time and place of the epidemics of miliary fever and cholera.* The interest attaching to this fact has been materially increased by some recent information about certain forms of disease described under the names of "sweating sickness" and "cholera sudoral," which point to a degree of connexion—extrinsic or intrinsic—between the two diseases.

At the first outbreak of cholera in France in 1832, attention had been drawn to that coincidence, the more so that miliary fever then became generally diffused after a quiescent period of eleven years, and began to spread simultaneously with cholera in the departments of the Oise,[1] Seine-et-Oise,[2] and Pas-de-Calais.[3] The same fact was observed over a still larger area in the second epidemic of cholera in France in 1849; miliary fever then became again widely prevalent in the most diverse parts of the country, and there are many records showing the coincidence of the two diseases in time and place in the departments of the Marne,[4] Seine-et-Marne,[5] Oise,[6] Seine-et-Oise,[7] Somme,[8] Yonne,[9] Puy-de-

[1] Menière, Hourmann.
[2] Dubun.
[3] Defrance
[4] Boinet (for the arrondissement of Epernay), Reveillé-Parise (for Fontenay).
[5] Gaultier.
[6] Verneuil, Tourrette.
[7] Bourgeois (for the arrondissement of Etampes).
[8] Foucart, Bucquoy.
[9] Lachaise, Badin et Sagot.

Dôme,[1] Gard,[2] and Hérault.[3] In the year 1853 the two diseases again occurred epidemically side by side[4] in the departments of Haute-Marne and Seine-et-Marne, and in like manner we find them closely associated in the widest diffusion in 1854 and 1855 in the departments of Haute-Sâone,[5] Vosges,[6] Haute-Marne,[7] Côte-d'Or,[8] Aube,[9] Haute-Garonne,[10] Hérault,[11] Jura,[12] Meurthe,[13] Charente,[14] Landes,[15] Basses-Pyrenées,[16] Hautes-Pyrenées,[17] and other departments.[18] Outside France, the same coincidence of cholera and miliary fever has not been observed, as far as I know, except in 1832, when, during the prevalence of cholera in Meiningen, miliary fever occurred in an adjoining village which was exempt from the cholera, and during the epidemics of 1849 and 1866 at many places in Belgium and Luxemburg.

The relation to one another of those diseases, when they were associated, has been found to be different in different epidemics. Very often the epidemic of miliary fever has preceded the outbreak of cholera, and has disappeared as the latter epidemic developed ; this happened in the departments of Côte-d'Or and Vosges,where, as Jacquot remarks, miliary fever was "an almost inseparable companion" of

[1] Nivet et Aguilhon.

[2] Gaultier (for Beaucaire).

[3] Arnaud (in 'Revue thérap. du Midi,' Oct., 1855, relating to Marseillan).

[4] Vergne.

[5] Bertrand.

[6] Destrem, Jacquot (especially for the arrondissements of Neufchâteau and Mirecourt, in the former of which 100 out of 132 communes were attacked by both diseases; in 94 communes about 19,000 persons suffered from miliary fever, at least 6000 from fully developed cholera, and 13,000 from cholerine).

[7] Jacquot, Barth.

[8] Clausse (for La Manche), Dechambre.

[9] Hullin.

[10] Millon (in 'Journ. de Méd. de Toulouse,' October, 1855, for Revel).

[11] Arnaud (l. c., for Marseillan), Saurel (in 'Revue thérap. du Midi,' Sept., 1855, for Marviel), Barth (for the arrondissement of Bézieres).

[12] Dechambre.

[13] Barth (for Houssonville, arrondissement of Luneville).

[14] Barth (for Cognac).

[15] Barth.

[16] Micé ('Journ. de Méd. de Bordeaux,' Dec., 1855), and Rossoutrot (for the arrondissement of Bayonne).

[17] Barth.

[18] See also the account by Fièvet (in the 'Gaz. des Hôpit.,' 1854, No. 107).

cholera. At other times both diseases have broken out nearly simultaneously, have run the same course, and gone away together. Sometimes miliary fever, having been the forerunner of cholera, has continued during the epidemic of the latter, and even survived it. More rarely miliary fever has come in the train of cholera epidemics. Lastly, both diseases have been prevalent at the same time in districts directly adjoining, in such a manner that the epidemic prevalence of the one disease has excluded the other; thus, in the department of Côte-d'Or, at the village of La Marche with 2000 inhabitants, there were 97 cases of miliary fever and only 43 cases of cholera, while at Flammerans, adjoining, a considerable diffusion of cholera took place, with only scattered cases of miliary fever.

Along with this coincidence in time of epidemics of miliary fever and cholera, a coincidence or combination of the two diseases has not unfrequently been observed in one and the same individual. Most usually this combination has found expression in a peculiar conformation of the miliary fever, choleraic diarrhœa and other symptoms of cholerine being added thereto;[1] or, miliary fever has developed itself in the course of cholerine, in such a way that the characteristic intestinal dejecta, the præcordial anxiety, the muscular cramps, and the like, have given way before an outbreak of copious perspiration, and the miliary fever has thereafter pursued its regular course (Verneuil). These cases, in the unanimous opinion of observers, always ended favorably, only that convalescence was in most of them remarkably protracted and interrupted by various obstacles, wherein they contrasted in a striking manner with the relatively mild course of the simple disease. It happened much more rarely that cholera developed in an individual ill with miliary fever; this has been either at the very commencement of the disease, in which case the sweating ceased, and there occurred cramps and collapse, and usually very soon death; or, more commonly towards the end of the miliary fever or in the period of convalescence, when the incident has mostly led to a bad prognosis.[2]

[1] Menière, Foucart, Bucquoy, Verneuil, Jacquot, Dechambre, Badin et Sagot, Defrance, Colson, Lachaise, Arnaut, Miecé, Rossoutrot, and others.

[2] Menière, Hourmann, Barth (for Cognac), Boinet, Badin et Sagot, Vergne,

Lastly, it has been very seldom[1] that miliary fever has appeared during the course of cholera (Dechambre), or in the stage of reaction, or during the convalescence from it (Micé), and the complication has, for the most part, brought no real danger to the patient (Dechambre).

In view of the facts here adduced, there appears to me to be no question at all that the coincidence of miliary fever and cholera, in the epidemic and in the individual, does not rest upon mere chance, but that there is a certain connexion between the two diseases under the circumstances mentioned. We find still further support for this view in the latest records of pestilence, wherein we meet with peculiarly-constituted forms of sweating sickness observed at various parts of the globe, always in the company of cholera and partly even sharing in the phenomena of that disease. These observations form an interesting complement to the history of the sweating diseases.

The first account of the form of disease in question occurs in Murray's[2] report on a malady described by him under the name of "sweating sickness," which he had observed in June, July, September, and October, 1839, and in June and July, 1840, at Mhow (in Malwa), where he was stationed. Cholera was epidemic in the neighbourhood, but it occurred only in sporadic cases at Mhow itself, while malarial fever was prevalent there during the autumn at the same time as the sweating disease.

The disease announced its attack with a prodromal stage of several days, during which the patients, besides complaining of slight headache and pressure in the region of the stomach, suffered from loss of appetite and sleeplessness, with watery stools several times a day, while the heart's action appeared to be notably weakened. Chills, with heats following, marked the onset of the developed disease, and at the same time there was an increase of the headache and of the associated symptoms before mentioned; the patients complained of extreme exhaustion, acute pain in the præcordial region, and thirst, and they were very restless. Next there occurred watery discharges from the bowel, with little colour, sometimes also vomiting of similar matters, cramp in the extremities, want of breath, and a feeling of obstruction and

[1] Boinet states that he has never seen this kind of combination, and many other observers consider it as rare (Dechambre).

[2] 'Madras Quarterly Med. Journ.,' 1840, ii, p. 77, and 1841, iii, p. 80. This periodical being rare in Europe, I consider a somewhat detailed account of this interesting observation to be called for here.

anxiety in the præcordia; the pulse was small and quick, the heart's impulse became imperceptible, and the skin was covered with running sweat. In the worst cases, all these troubles disappeared except the intense thirst, the oppression in the chest, and the profuse sweating; but gradually the pulse became imperceptible, a comatose condition took the place of consciousness hitherto unimpaired, and death occurred often within ten hours of the onset. Vomiting and cramps were not specially prominent or constant phenomena during the course of the disease, while, on the other hand, complete retention of urine and absence of bile in the stools were always observed. When the course of the disease was favorable, the pulse became fuller and slower, the feeling of burning and oppression in the præcordia went off, the dejecta became feculent and tinged with bile, the patient passed large quantities of urine, and fell asleep to awake quite well; or, in other cases less favorable and less prompt, the fever and running sweats persisted some time longer. The same series of phenomena often recurred after twelve to forty-eight hours; not unfrequently there were even several onsets one after the other which, in the favorable cases, became more and more slight; while in unfavorable cases the coma increased, lasted longer and longer, and the disease ended in death. But recovery might take place even in the most severe cases, and Murray saw one patient recover after lying comatose for three days. Convalescence was always marked by a great degree of weakness; the patients, while getting well, often complained for a long time of an oppressive feeling about the heart, and not unfrequently there were relapses and recurrences. At the anatomical examination of two individuals who had died of this disease, Murray found a remarkably dark and watery state of the blood, serous effusion into the membranes of the brain, and much accumulation of blood in the thoracic and abdominal organs; there were no important anatomical changes besides. He believed that death took place from uræmia, in consequence of the complete suppression of the urinary secretion which always occurred in the course of the disease. In the disease itself he discovers the type of miliary fever; but there was at the same time one series of phenomena that proved unmistakeably, as he thinks, the near relations of the disease to cholera, while another showed its affinities to malarial fever.

Connecting naturally with this narration, are certain accounts of a form of disease observed on European soil at a time when cholera was epidemically prevalent; if it be not completely identical with the Indian sweating disease, it resembles it in a high degree, and shows, like it, a very close relation to miliary fever. Roux, a navy surgeon at Toulon, was the first to call attention to this peculiar sickness, and he gives a sketch of it [1] under the name of "choléra

[1] 'Union méd.,' 1855, Nos. 27—32, 1857, Nos. 131, 139, 142, 143.

cutané ou sudoral" based upon observations made by himself at that city in the cholera years, 1849, 1854, and 1855; he touches also upon the accounts of other French naval surgeons[1] who had observed the disease on board the French fleet in the Black Sea during the Crimean war at the time of the cholera epidemic of 1854. These accounts have been subsequently amplified; not to mention the less complete data furnished by several other French physicians, there have been communications by Houlés,[2] Bourgogne,[3] and Lespinois[4] on the occurrence of the same form of disease at the time of the cholera epidemic of 1854 in Languedoc (particularly in Sorèze, Revel, and other places in the department of Tarn) and in Condé (Nord).

According to the description given by Roux, this disease, distinguishable from the cholera then prevalent by the profuse sweats, the absence or scantiness of the discharges, whether vomit or stools, as well as by the frequent relapses and by its long duration, had usually a sudden onset, and that was especially often in the night; the patient was overcome by a feeling of extreme weakness resembling a swoon, and at the same time his face became pale, he felt cold, the voice changed, the pulse became slower, and sometimes also there was sickness and a desire to go to stool. When this painful condition had lasted a short time, or even several hours, a reaction set in; the pulse rose, the body became warm, and there now broke out an inexhaustible ("intarissable") sweat which ran like a stream day and night, so that the patient was obliged to have the bed linen continually changed. Gradually the perspiration and heat declined, and the patient now lay in a state of extreme exhaustion, the features sunken and of an earthy pallor, the extremities as if broken or seized with cramp, appetite and sleep destroyed, while there was a particularly troublesome sense of pressure and a feeling of obstruction at the epigastrium. After a few days, appetite, sleep, and strength returned, and the patient seemed to be in full convalescence, when suddenly a fresh onset occurred with the whole chain of symptoms as before, and these relapses would recur three, five, or six times, and even oftener, and at intervals either regular or irregular. The disease was observed only in adults, it ran an entirely favorable course, and even the manifold troubles that remained behind, such as the neuralgias, the feeling of oppression about the heart, the digestive disturbances, the capricious temper, and the like, went away com-

[1] Ibid., 1855, No. 31.

[2] 'Revue méd.,' 1855, August, September.

[3] 'Lettre sur le traitement abortif du Choléra Asiatique,' Valenciennes, 1854, and in the 'Annal. de la Soc. Méd. de Bruges,' 1860, Nov., Dec.

[4] 'Essai sur la Cholera cutané ou sudoral,' Montpellier, 1868.

pletely in course of time.[1] The disease on board the French fleet in the Black Sea, according to the descriptions of Beau, and that in Condé according to Bourgogne, and in the department of Gard according to Houlés, assumed a similar form, but it wanted the peculiar intermissions, and was much more malignant; it usually ran a fatal course with pronounced symptoms of asphyxial cholera, excepting that profuse sweats took the place of the intestinal transudation. "In 361 patients," we read in Beau's account, "there were present all the most marked symptoms of algid cholera: cyanosis, icy coldness of the extremities and of the tongue, cold sweats running over the whole cutaneous surface, in such quantity as to soak the linen of the patients in a few minutes and to macerate their epidermis; absence of pulse at the wrist, extinction of the voice, rapid wasting, typical facies choleraica, very painful cramps, sometimes suppression of urine. The characteristic vomiting and diarrhœa were all that was wanting in many cases; and therein lies one of the marked peculiarities of our epidemic. *The copious diaphoresis which we have dwelt upon appears to take the place of the usual hypersecretion from the intestine.*"

If we now put together the facts stated in the preceding pages, with a view to forming an opinion on the nature of the disease-forms here described under the general designation of "sweating diseases," we shall find that one constant phenomenon, characteristic of and peculiar to all those diseases, is the morbidly increased activity of the skin, partly taking the form of an abnormal transudative process, partly breaking out in exanthems; we see also that these pathological processes are accompanied by decided indications of a profoundly disturbed circulation and nutrition, either caused by an affection of the nervous system, or at least accompanied by such an affection. So far from being able to assign a *critical* significance to the perspiration and exanthem which occur in the miliary febrile diseases in question, we must judge of the nature and character of these symptoms from the same point of view as we judge of other abnormal processes of transudation due to similar disturbances of a general kind. None of the processes of disease that are accurately known to us offers so striking an analogy in this respect, as the disease to which sweating sickness shows unmistakeable relations in another direction, viz. *the cholera;* and this analogy is so strong that on the first appearance of cholera

[1] It will be seen that this was not, as might be surmised, a form of pernicious malarial fever, partly from the fact that quinine was completely powerless in the disease, and also from the facts stated in the sequel.

upon European soil, Hufeland drew attention to the resemblance between it and the oldest form of sweating disease known to us, the sweating sickness of the English. The French observers, who have so often and so uniformly observed the epidemics of miliary fever to coincide in time with cholera, have identified the two diseases from the genetic point of view, cholera being taken to be a kind of internal miliary fever ("Comme une sorte de suette interne," as Dubun expresses it), and miliary fever a kind of "cholera of the skin"; and they think that they have found material support for this idea in the hybrid form of cholera and miliary fever above described.

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CHAPTER IV.

SMALLPOX.

§ 34. Its Antiquity and Original Habitat.

Few points in pathology have occupied the attention of students of the history of medicine more than the question of the origin and antiquity of the smallpox. In the sixteenth century a keen discussion had already begun as to whether that disease had been known to the physicians of antiquity; in the two following centuries, and down even to the most recent times, the same question has been the subject of numerous learned controversies, without anything better than guess-work resulting from all these inquiries.[1] The first unambiguous statements about the disease from a medical source occur in the well-known treatise of the tenth century by Rhazes, 'De Variolis et morbillis,'[2] in which it is taken as established that Galen was acquainted with the disease, and fragments given from the 'Pandects' of the Alexandrian physician Ahron, dating from the fifth or sixth century, from which it follows that he was well acquainted with the smallpox.[3] Rhazes speaks of the smallpox as a disease generally known over the East, and the same opinion is expressed by subse-

[1] Among the writers on this subject most worthy of mention are: Hahn, 'Variolarum antiquitates,' Brigæ, 1833, and 'Carbo pestilens a carbunculis distinctus,' Vratislav, 1736; Werlhof, 'Disqu. de Variolis et anthracibus,' Hannov., 1735, in 'Ejd. Opp.,' Hannov., 1755, ii, 469; Moore, 'History of the Smallpox,' Lond., 1802; Willan, 'Miscellaneous Works,' Lond., 1821; Krause, 'Ueber das Alter der Menschenpocken,' &c., Hannov., 1825; Häser, 'Lehrb. der Gesch. der Med.' (1876), iii, 18–59 (very thorough inquiry).

[2] In Arabic with Latin translation by Channing, Lond., 1776; another Latin version in Mead., 'Opp.' Neapol, 1752, p. 51. [Eng. transl. by Greenhill, Sydenham Soc., Lond., 1848.]

[3] In the treatise above mentioned; also in 'Continens Brix.,' 1486.

quent writers, by Ali Abbas,[1] Avicenna,[2] and other Arabian physicians of the tenth and eleventh centuries, as well as by Constantinus,[3] who had been educated in Arabian schools; so that the fact, otherwise well corroborated, of the general prevalence of smallpox during the splendour of the Arabian power, not only in eastern but in western lands as well, is placed beyond all doubt. It is more difficult to decide when and by what road the disease came to Arabia. Reiske[4] quotes the following passage from Massudi's 'Golden Mead:' "Hoc demum anno [*i.e.* in the second year of the siege of Mecca by the Abyssinians during the so-called Elephant War, or about the year 370 A.D.] comparuerunt primum in terris Arabum variolæ et morbilli, quorum quidem aliqua fuerunt jam antea inter Israelitas, non tamen Arabum terras invaserunt, nisi tum demum;" and a confirmation of this statement occurs in the writings of El-Hamisy,[5] and in other Arabian chroniclers.[6]

Probably smallpox is the disease here referred to, and it is possible that the outbreak of it was connected with the invasion by the Abyssinians; while the assertion that this was the first occurrence of the disease in Arabia can neither be made good nor set aside. But it would be more than seems warranted if we were to conclude from this that the original habitat of the disease has to be assigned to African soil; for there are other and not less reliable sources of information which make out smallpox to have been prevalent much earlier in Asiatic countries, and to have been generally diffused in that continent at a time when it was hardly credible that the disease should have been imported from Africa. Holwell, who was for a long time resident in India, and whose tes-

[1] 'Regalis dispositionis Theorices,' lib. viii, cap. 14; 'Practices,' lib. iv, cap. 5, Lugd., 1523, fol. 97, 198.

[2] 'Canon,' lib. iv, Fen. 1, tract. iv, cap. 6 ff., Venet., 1564, ii, 71.

[3] 'De morbor. cognitione,' lib. viii, cap. 8, Opp. Basil. 1536, 152.

[4] 'Opuscul. med. ex monumentis Arabum,' Hal., 1776, 8.

[5] 'According to Bruce, 'Travels to the Sources of the Nile,' Lond., 1790, i, 516. The chronicler narrates that flocks of strange birds (Ababil, the Persian term for smallpox) came over the sea to Mecca, each one carrying in its beak and in its claws stones as large as a pea, which they let fall upon the Abyssinians, so that their armour was pierced and the whole army slain; this was the time, the narrator adds, when smallpox and measles first broke out in Arabia.

[6] See also Häser, l. c., p. 59.

timony is deserving of all credit, states[1] that immemorial traditions have existed in the Brahmin caste concerning the prevalence of smallpox in India, that there has existed there since the earliest times the temple-worship of a deity whose protection and help was invoked on the epidemic outbreak of the disease,[2] and that there is contained in the Atharva Veda a description of this temple service together with the prayers used by the Brahmins at the inoculation with smallpox, which has been practiced there from time immemorial. Wise,[3] who gives a sketch from the oldest Sanscrit writings on medicine (the Charika and Susruta) of the spotted and pustular skin diseases, and among them of the smallpox, is convinced that this disease had been prevalent there at a very remote period; and he makes the conjecture, which is, however, by no means well founded, that it came from India to the western parts of Asia, and thence to European and to African soil. An equally great antiquity has been claimed by Moore[4] for the diffusion of smallpox in China, on the authority of a treatise with the title, 'Heart Words on the Smallpox,' which had been edited by the Imperial College of Physicians; this treatise is based upon the oldest writings of Chinese physicians, and in it the first appearance of the disease in China is referred to the time of the Tsche-u dynasty, or the period between 1122 and 249 B.C. According to Smith's researches,[5] the disease appeared for the first time in that country during the dynasty of Han (206 B.C. to 220 A.D.) having been imported, as he adds, from Central Asia or from countries to the south-west, perhaps, therefore, from India. Both statements may be easily reconciled if we assume that the malady first appeared in the third century B.C., an assumption which agrees with the statement

[1] 'Account of the Manner of Inoculating for the Smallpox in the East Indies,' Lond., 1767, p. 8.

[2] This temple-worship is very widely spread in India; the goddess to whom it is offered, bears various names in various parts of the country, corresponding mostly, as it seems, to the character of the disease or to the mode of treatment. According to Moore ('Med. Times and Gazette,' 1869, Nov., p. 634), numerous temples of that kind are met with in Rajpootana.

[3] 'Commentary on the Hindu System of Medicine,' Lond., 1860, p. 233.

[4] L. c., p. 21.

[5] 'Medical Times and Gazette,' 1871, Sept., p. 277.

of Lagarde,[1] who puts the antiquity of smallpox in China at upwards of 2000 years.

The question of the antiquity of smallpox in African countries is entirely beyond our answering; nor on European soil can the occurrence of the disease be traced back with certainty beyond the Christian era.[2] The first references to smallpox for these countries, that are at all reliable, are met with in the fragments (preserved by Aetius)[3] by the physician Herodotus, who lived at Rome in the time of Trajan; next come the accounts given by Galen[4] of the pestilences throughout the whole Roman empire of the east and west, in the time of Antoninus (A.D. 160—168); then the description by Marius of Avenches[5] of the epidemic that was widely prevalent in France and Italy in 570, wherein the name "variola" occurs for the first time as a designation of the disease; and finally, there is the account by Gregory of Tours[6] of the sickness that overran a great part of Southern Europe in 580. Entirely unambiguous statements as to the prevalence of smallpox in Europe date from the eleventh and twelfth centuries; and it is by no means improbable, as many chroniclers of that time agree in saying, that the tumultuous popular movements during the crusades contributed materially to the general diffusion of the disease. Almost all the medical writers of the middle ages mention the smallpox,[7] while some of them, such as Gordon,[8] Varignana,[9] John of Gaddesden,[10] Bertuc-

[1] 'Arch. de méd navale,' 1864, March, p. 190.

[2] There is little foundation for the opinion first expressed by Krause (op. cit., p. 50), and adopted by Daremberg (in Prus, 'Rapport sur la peste et les quarantaines,' Paris, 1846, p. 238), and by Littré ('Oeuvres complètes d'Hippocrate,' v, p. 48), that the Plague of Athens during the Peloponesian war, B.C. 428, as described by Thucydides, was an epidemic of smallpox.

[3] Lib. v, cap. 130, ed. lat., Basil, 1535, i, p. 226.

[4] A complete collection of all the passages in the writings of Galen which bear on the question will be found in Hecker's 'De peste Antoniniana commentatio,' Berol, 1835; and in his 'Wissenschaftliche Annalen des Ges. Heilkd.,' 1835, xxxii, 1.

[5] 'Chronicon' in Bouquet's 'Collection des historiens de France,' Paris, 1738, ii, p. 18.

[6] 'Historia Francorum,' vi, cap. 14, Parisiis, 1610, p. 263.

[7] Gruner ('De variolis et morbillis fragmenta medicorum Arabistarum,' Jen. 1790) has published a tolerably complete collection of the more important accounts of smallpox in the writings of mediæval physicians. A copy of this extremely rare work is in my possession.

[8] 'Lilium medicinæ,' pars. i, cap. 12, Lugd., 1574, p. 51.

[9] 'Secreta sublimia,' tract. ii, cap. 1, Lugd., 1526, fol. 43. *b*.

[10] 'Rosa anglica,' Aug. Vindel, 1594, p. 1041.

cio,[1] Gentilis de Fuligno,[2] Valescus de Tharanta,[3] Concorregio,[4] Antonio de Gradis,[5] and Blasius Astarius,[6] treat the subject at greater length, but still in the spirit of their Arabian models, and entirely without regard to the epidemiological aspect of the disease. Consequently for the purpose of our epidemiological inquiry, we have to fall back upon the mediæval chroniclers; but in them too there is very little to be found, nothing, indeed, that justifies more than a conjecture that the smallpox played at any rate a prominent part among the pestilences of the middle ages.

But, although the origin of smallpox still remains an unsolved problem, and its primæval history is still shrouded in complete obscurity, so much, at least, appears to be made out for certain, that this malady, like the plague, yellow fever, and other severe infective diseases, has its habitat at only one or two points on the globe, and that its diffusion over the earth's surface has been brought about by successive importations of the morbid poison from those original seats. The *native foci of smallpox* may be looked for in *India* and in the *countries of Central Africa*. As we shall see in the sequel, it is not possible to make out clearly, except in a few instances, at what times the disease came from those centres to the several regions of the Eastern and Western Hemispheres. At the present time the dominion of smallpox extends over almost the whole inhabited globe, and there are only a few regions, as we shall see in the following account of its geographical distribution, that still enjoy a complete immunity from it.

Although the introduction of vaccination into the more civilised states has greatly limited the amount of the disease, still the area of its distribution has undergone no real curtailment, and even at the present day smallpox remains one of the most widely spread of the acute infective diseases.

[1] 'Collectorium totius med.,' lib. ii, tract. i, cap. 16, Lugd., 1509, fol. 274. (This author has not found a place in Gruner's collection).

[2] 'De febribus,' tract. iv, cap. 4, Venet., 1503, p. 85.

[3] 'Philonium,' lib. vii, cap. 17, Lugd., 1490, fol. 326.

[4] 'Practica de curis febrium,' Venet., 1521, fol. 93, *b*.

[5] 'De febribus,' cap. 26, in 'Clementii Clementini Lucubrationes,' Basil (1535), p. 279.

[6] 'De curis febrium,' in Gatinaria, 'De curis ægritudinum,' Lugd., 1525, fol. 75, *b*.

§ 35. Geographical Distribution.

One of the most intense foci of smallpox is met with on *African* soil, in the countries of the Nile basin, *Egypt, Nubia, Kordofan,* and the *Abyssinian* Highlands. "It increases in frequency and severity," says Pruner,[1] referring to these countries, "as we penetrate into the interior of this part of the world, or, in other words, as we ascend the Nile; it appears to be the one great sickness there." From the interior of Abyssinia it is often imported to the coast,[2] so that, as Courbon states,[3] one seldom finds an Abyssinian without the marks of smallpox. According to verbal statements made by Dr. Arnaud to Pruner, the Shillook country forms the southern boundary of this focus of smallpox, but there is reason to think that the endemic area of the disease reaches much farther, that it extends, in fact, to the interior parts of South Africa. One fact in support of this, given by Lostalot-Bachoué,[4] is that smallpox is permanently active on the *Zanzibar Coast* and on the *East Coast of Africa* in general; and another is that it never ceases its ravages in *Madagascar,*[5] where an attempt has been made to introduce vaccination since the severe epidemics of 1866-67. Again, the frequent prevalence of smallpox among the natives of *Cape Colony* points to a central focus of disease in the interior of *South Africa.* Lichtenstein, who travelled through Kaffirland in 1804, and found many of the natives pitted from the smallpox, became persuaded on penetrating farther into the interior, that the malady was widely diffused over the whole continent, while

[1] 'Die Krankheiten des Orients,' Erlangen, 1847, p. 127. See also Hartmann, 'Naturgeschichtlich-med. Skizzen der Nilländer,' Berl., 1865. Of its endemic occurrence in Kordofan there is an account by the physician Ebn-Omar-el-Jounsy, 'Voyage au Darfur,' Paris, 1845; for Abyssinia (Shoa), by Rochet d'Héricourt, 'Voyage dans le pays d'Adel,' Paris, 1841, p. 308.

[2] Martin, in the 'Lancet,' 1869, Jan., p. 140.

[3] 'Observ. topogr. et méd. rec. dans un voyage à l'isthme de Suez,' Paris, 1861, p. 30.

[4] Étude sur la constitution phys. et méd. de l'île de Zanzibar,' Paris, 1876, p. 47.

[5] Davidson, in the 'Med. Times and Gaz.,' 1868, Dec., p. 646; Borchgrevink, in 'Norsk Mag. for Laegevidensk,' 1872, iii. Raekke ii, p. 247.

he did not find the remotest reason for believing that it had been imported from the coast. "All that I could learn on that subject," he says,[1] "served more and more to confirm the opinion of the Kaffirs that this disease is indigenous in Africa. The history of this people does not, indeed, go far back into antiquity; but all the best informed persons among them were of one mind, that the malady had been prevalent in their midst as long as they had been a people at all. There could hardly be in their case any question of the disease having been communicated by Europeans; for they dwell so far from the coast that even so recently as ten or twelve years ago the tales of a great water (the sea), and of white men, were counted among their fabulous legends, and did not find credence until the arrival of some Dutch travellers from Cape Colony. On the other hand, they told us of the Macquini (?), a great nation living far to the north, in the very heart of unexplored tropical Africa, from whom they had got their last epidemic of smallpox, transmitted through the tribes occupying the country between."[2] The disease was imported to *Cape Colony*, according to Murray,[3] for the first time in 1713 by a ship from India, next in 1755 from Ceylon, and afterwards in 1812 by a slave ship from Mozambique, from which date down to 1840 the Colony remained free from the disease.[4] [Cape Town and the country round about were visited by a very severe epidemic in 1882, with a high mortality.]

In *Réunion* the smallpox became prevalent for the first time in 1729, having been introduced from Madagascar; the next outbreak took place in 1827, again in consequence of importation of the disease by a slave ship, to which source the later outbreaks in 1850 and 1858 are also traceable. In the intervals of these several epidemics, sporadic cases of

[1] 'Hufeland's Journal der Heilkd.,' 1810, xxxi, pt. i, p. 1. See also Fritsch, 'Archiv für Anatomie und Phys.,' 1867, p. 738.

[2] Scherzer ('Zeitschr. der Wiener Aerzte,' 1858, No. 11, p. 166) calls attention to the fact that inoculation is generally practised among the Hottentots, the Dutch having introduced it, and that that race are much less apt to suffer from smallpox than the Kaffirs, who shun the practice, as they do also vaccination, and who often have the whole population of a village swept away by the smallpox.

[3] 'Lond. Med. Gaz.,' Dec., 1833; Oct., 1834.

[4] 'Zeitschrift der Wiener Aerzte,' 1858, No. 40, p. 630.

smallpox have never been observed.[1] *Mauritius*, according to Charpentier, has been often visited by epidemics of the disease under the same circumstances, without any sporadic cases occurring in the intervals. *St. Helena* had remained quite exempt down to 1836;[2] I have not ascertained whether this immunity has continued. There is no information forthcoming as to the occurrence of smallpox along the southern part of the *West Coast of Africa*. On the *Guinea Coast*, according to the unanimous opinion of observers,[3] the disease is not indigenous; it occurs from time to time as an epidemic, sometimes so disastrously that whole villages are ravaged by it. The same is true for the coast of *Senegambia*,[4] where the endemic prevalence of the disease is absolutely denied by Gauthier,[5] and for the adjoining regions of *Soudan*,[6] as well as the coast of the *Barbary States*, of *Tunis*,[7] and of *Algiers*.[8] The locality most affected in the last mentioned is Kabylia, where Claudot,[9] confirming Challan,[10] speaks of smallpox as the greatest scourge of the country. From the west coast, smallpox has been repeatedly

[1] Azéma, in the 'Arch. gén. de méd.,' May, 1863. See also Follet, in the 'Revue méd.,' Dec., 1834, p. 440.

[2] McRitchie, in the 'Calcutta Med. Transact.,' 1836, viii, App. xxix.

[3] Compare the accounts by Monnerot ('Considér. sur les maladies endémiques . . . . du Gaboun,' &c., Montpellier, 1868, p. 40) for the Gaboon country; for the Benin coast by Daniell ('Sketches of the med. topogr. . . . of the Gulf of Guinea,' Lond., 1849, p. 48), and by Hewan ('Lancet,' 1877, Sept., p. 388), who states in an account of the severe smallpox epidemic of 1869 in Old Calabar, that the disease had not been seen in Benin for a space of eighteen years; further, by Moriarty ('Med. Times and Gaz.,' 1866, Dec., p. 662) for the Gold Coast, and by Boyle ('Med.-Histor. Account of the Western Coast of Africa,' Lond., 1831, p. 400), Gordon ('Edin. Med. Journ.,' 1856, Dec.), and Clarke ('Trans. of the Epidemical Soc.,' 1860, i, p. 102) for the coast of Sierra Leone.

[4] Thevenot, 'Traité des malad. des Européens dans les pays chauds,' Paris, 1840, p. 249; Thaly, in 'Arch. de méd. nav.,' 1867, Sept., p. 174; Berger, 'Considér. hyg. sur le bataillon de tirailleurs Sénégalais,' Montp., 1868, p. 53.

[5] 'Des Endémies au Sénégal,' Paris, 1865. p. 18.

[6] Quintin, who lived for two years in Segu, the capital of the kingdom of Bambarra, where no European had penetrated up to 1864, states that not a single case of smallpox occurred during that period. There, also, the disease breaks out now and then in disastrous epidemics ('Extrait d'un Voyage dans le Soudan,' Paris, 1869, p. 37).

[7] Ferrini, 'Saggio sul clima e sulle malattie dell'regno di Tunisi,' Milano, 1860, p. 151.

[8] Bertherand, 'Médecine et hyg. des Arabes,' Paris, 1855.

[9] 'Rec. de mém. de méd. milit., 1877, p. 193.

[10] 'Gaz. méd. de l'Algérie,' 1868, p. 115.

introduced into the *Cape Verd Islands*[1] and into the *Canaries*.[2] In *Asia Minor*,[3] *Syria*,[4] and *Mesopotamia*,[5] where they have not succeeded hitherto in introducing vaccination as a general practice in place of inoculation, smallpox still plays as prominent a part in the sickness and mortality as it used to do. This is true also of *Persia*[6] and *Arabia*,[7] and in a still higher degree of *India* and *Further India*, where the disease is thoroughly endemic over large tracts of country. Pringle, basing upon thirteen years' medical experiences in India, speaks of smallpox as the severest scourge of the country;[8] if cholera, he says, carries off hundreds every year, if the victims of famine are to be counted by thousands, these are but infinitesimal quantities beside the frightful amount of devastation caused in India by the smallpox. In the years from 1866 to 1869, in the Presidencies of Bombay and Bengal with a total population of forty millions in round numbers, 140,000 persons died of the disease;[9] in the years 1875 and 1876 the mortality from this disease in the whole of India amounted to 200,000, and in the two preceding years to 500,000.[10] Among the regions of India most severely visited are many parts of the Presidency of Bengal,[11] particularly the Province of Orissa[12] and the southern slopes of the Himalaya;[13] in the Madras Presidency, the district of Madras,[14] Pondicherry,[15] the Malabar coast,[16] par-

[1] Hopffer, in 'Arch. de méd. nav.,' 1877, March, p. 161.

[2] Busto y Blanco, 'Topogr. med. de las islas Canarias,' Sevilla, 1864.

[3] West, in 'New York Med. Record,' 1869, March, iv, p. 27.

[4] Robertson, 'Edin. Med. and Surg. Journ.,' 1843, July, p. 58; Guys, 'Statist. du Paschalik d'Alep.,' Marseille, 1853, p. 63.

[5] Ffloyd, in 'Lancet,' 1843, July; Evatt, 'Army Med. Rep.,' 1874, xvi, p. 178.

[6] Polak, in 'Wochenbl. der Wiener Aerzte,' 1857, No. 44, p. 709.

[7] Palgrave, in 'Union méd.,' 1866, No. 20, p. 308.

[8] 'Lancet,' 1869, Jan., p. 44.

[9] Cornish, ib., 1871, May, p. 703.

[10] Murray, ib., 1878, Nov., p. 699.

[11] See Twining, 'Clinical Illustr. of the Diseases of Bengal,' Calcutta, 1835, ii, p. 432; 'Report of Smallpox Commissioner,' Calcutta, 1850; Milroy, in 'Transact. of the Epidemiol. Soc.,' 1865, ii, p. 153.

[12] Shortt, in 'Indian Annals of Med. Sc.,' 1858, July, p. 505.

[13] Curran, in 'Dubl. Quart. Journ. of Med. Sc.,' 1871, Aug., p. 101.

[14] Cornish, in 'Madras Quart. Journ. of Med. Sc.,' 1861, July, p. 84; Shortt, ib., 1866, July.

[15] Huillet, in 'Arch. de méd. Nav.,' 1867, Dec., p. 419.

[16] Cleveland, in 'Madras Quart. Journ. of Med. Sc.,' 1863, Jan., p. 32.

ticularly Cochin;[1] in the Bombay Presidency, the districts of Gujerat and Upper Sind;[2] many regions of the North West Provinces,[3] and especially the Punjaub, where, according to the statement of De Renzy,[4] smallpox counts among the endemic diseases: in Lahore alone, 7000 persons died of it in 1865 in the space of two months.[5] In the districts more remote from general traffic, such as the Nilghirri Hills, the disease appears only at long intervals, but when it does come it brings a disastrous epidemic.[6] Similar accounts of the severe ravages of the disease come from those parts of *Lower India* that we know most of as regards things medical, such as Burmah,[7] the peninsula of Malacca,[8] and Cochin China,[9] as well as from many parts of the *Indian Archipelago*, —Borneo,[10] Timor,[11] Amboina,[12] Ternate,[13] the Nicobars,[14] and other islands. Within the last ten years, the Dutch Government appear to have succeeded in introducing vaccination more generally and in limiting the disease proportionately. This has been still more the case in *Ceylon*, where vaccination was introduced by the English authorities as early as the beginning of the century; so that Davy,[15] writing in 1821, was able to speak of smallpox as almost completely exterminated, while later observers[16] have pointed

[1] Day, ib., 1861, Oct., p. 213.

[2] Don, in 'Bombay Med. Transact.,' 1840, iii, p. 10.

[3] McGregor, 'Observ. on the Principal Diseases in the N.W. Provinces of India,' Calcutta, 1843, p. 207.

[4] 'Brit. Med. Journ.,' 1874, Sept., p. 269.

[5] Account in 'Philad. Med. News,' 1865, p. 63.

[6] Young, in 'Calcutta Med. Transact.,' 1829, iv, p. 60; Mackay, in 'Madras Quart. Journ. of Med. Sc.,' 1861, July, p. 26.

[7] Dawson, in 'Philadel. Med. Examiner,' 1852, May.

[8] Ward, in 'Edin. Med. and Surg. Journ.,' 1831, July, p. 188; Dick, in 'Army Med. Report,' 1873, xv, p. 329.

[9] Richaud, 'Arch. de méd. nav.,' 1864, May, p. 356; Thil, 'Remarques sur les Maladies de la Cochin-Chine,' Paris, 1866, p. 33.

[10] Account in 'Arch. de méd. nav.,' 1872, Jan., p. 10; Bulwer, in 'Brit. Med. Journ.,' 1874, May, p. 618. In the Brunei country, with 30,000 to 40,000 native inhabitants, 4000 died of the disease within three months.

[11] Account in 'Arch. de méd. nav.,' 1870, July, p. 15.

[12] Ib., 1869, Sept., p. 177.

[13] Ib., 1870, March, p. 176.

[14] Steen-Bille, 'Reise der Corvette "Galatea" um die Welt,' Leipz., 1852, i, p. 244.

[15] Davy, 'Account of the Interior of Ceylon,' London, 1821.

[16] Kinnis, 'Letter on the Advantage of Vaccination, &c.,' Calcutta, 1837; Milroy, in 'Transact. of the Epidemiol. Soc.,' 1865, ii, p. 153.

out that the island is much better off as regards smallpox than the mainland adjoining. In *China* also, where vaccination was introduced in 1805,[1] a considerable decrease of the disease has shown itself at certain places,[2] whereas many other regions, including Chee-Foo, Shanghai, Peking, and the province of Fung-Thian-Foo (in southern Manchooria), where vaccination is very imperfectly practised and inoculation still in full repute, constitute permanent centres of the disease, and have often been ravaged by disastrous epidemics of it.[3] In *Corea*, Cheval found almost the whole population pock-pitted.[4] In *Japan*, where the smallpox is said[5] to have appeared for the first time in A.D. 736, having been introduced from Tartary, the efforts of the Dutch to introduce vaccination have had small success; according to all observers,[6] the disease is diffused everywhere and its ravages are often very great. Smallpox reached *Siberia* for the first time in 1630, from the nearest Russian province,[7] and spread thence with great rapidity to the Ostiaks, the Tunguses, the Yakuts, and the Samojeds, producing frightful havoc among them; in more recent times also, accounts reach us of the disastrous prevalence of the disease among the Samojeds.[8] *Kamschatka*, at the time when Müller travelled through it (beginning of 18th century), was quite free from the malady;[9] according to the account of Cook the disease gained admission there for the first time in 1767.

The continent of *Australia*, up to 1838, had enjoyed an absolute immunity from smallpox; towards the end of that

[1] Pearson, in 'Calcutta Med. Transact.,' 1833, vi, p. 361.

[2] Armand, in 'Gaz. méd. de Paris,' 1860, No. 17, p. 261; Friedel, 'Beiträge zur Kenntniss des Klimas und der Krankheiten Ost-Asiens,' Berlin, 1863, pp. 106, 122.

[3] Lagarde, in 'Arch. de méd. nav.,' 1864, March, p. 190; Cheval, 'Relat. d'une campagne . . . au Japon, en Chine, et en Corée,' Montpell., 1868, p. 41; Watson, in 'Edin. Med. Journ.,' 1869, Nov., p. 430; Morache, in 'Annal. d. hyg.,' 1870, Jan., p. 55; Dudgeon, in 'Glasg. Med. Journ.,' 1877, July, p. 320.

[4] L. c., p. 63.

[5] Schmid, in 'New York Med. Record,' 1869, Sept., p. 314.

[6] Friedel, l. c., p. 22; Account in 'Arch. de méd. nav.,' 1866, April, p. 278; Cheval, l. c., p. 31; Potocnik, in 'Arch. de méd. nav., 1875, Oct., p. 252; Wernich, in 'Deutsch. med. Wochenschr.,' 1878, No. 9, p. 101.

[7] Richter, 'Geschichte der Med. in Russland,' Moskau, 1817, iii, p. 313.

[8] Schrenk, 'Reise in die Tundren der Samojeden,' Dorpat, 1848, i, p. 546.

[9] 'Sammlung russischer Geschichte,' v, p. 74 (quoted by Richter, l. c.).

year, the disease appeared at Sydney,[1] having been imported probably from China; it lasted, however, only a short time, and remained absent from the continent until 1868. In that year it was introduced into Melbourne by a ship, and again it spread only to a slight extent and quickly died out.[2] By a rigorous inspection of ships on their arrival, it has been found possible to prevent subsequent importations, a notable instance of prevention having occurred in 1872.[3] *Tasmania* has hitherto quite escaped the disease;[4] so also has *New Zealand*, where an importation of it in 1872 was prevented by strictly isolating a vessel that had arrived with smallpox on board.[5] In many of the island groups of *Polynesia* the disease has been found to spread much more widely and to be much more destructive than in Australia. Its first appearance was in *Tahiti*, which has repeatedly since its discovery suffered from severe epidemics, although it has experienced somewhat less of the disease since the introduction of vaccination in 1843.[6] The *Hawaiian Islands* were visited by the smallpox first in 1853, when a ship brought it from San Francisco to Honolulu; in eight months the disease carried off 8 per cent. of the population,[7] and continued its ravages in the years following, so that the number of the inhabitants was much reduced;[9] another severe epidemic arose in 1872.[9] *New Caledonia* was quite exempt up to 1859,[10] when the disease was introduced and has not left the colony again.[11] The *Marquesas* were first visited by it in 1863;[12] the disease was brought to Noukahiva by natives returning, at the

[1] Account in 'London Med. Gaz.,' 1839, June, p. 477.

[2] Rochlitz, 'Archiv für Dermatologie,' 1872, iv, p. 395.

[3] Account in 'Med. Times and Gaz.,' 1872, Sept., p. 364.

[4] Millingen, in 'Calcutta Med. Trans.,' 1836, viii, App. xi; Hall, in 'Trans. of Epidemiol. Soc.,' 1865, ii, pp. 70, 293; Moore, 'Dubl. Journ. of Med. Sc.,' 1874, Feb., p. 151.

[5] Thomson, in 'Brit. and For. Med.-Chir. Rev.,' 1855, April; Bourse, 'Arch. de méd. nav.,' 1876, March, p. 179.

[6] Account in 'Arch. de méd. nav.,' 1865, Oct., p. 283.

[7] Gulick, in 'New York Journ. of Med.,' 1855, March.

[8] Le Roy, 'Relat.' méd. d'un voyage dans l'océan pacifique, &c.,' Paris, 1860.

[9] Account in 'Brit. Med. Journ.,' 1872, Oct., p. 474.

[10] Vinson, 'Topogr. méd. de la Nouvelle-Calédonie,' Paris, 1858.

[11] Charlopin, 'Notes rec. en Calédonie,' Montpell., 1868, p. 22.

[12] 'Lancet,' 1869, May, p. 599; Brunet, 'La race Polynésienne, &c.,' Paris, 1876, p. 34.

requisition of the French authorities, from the Chinca Islands, wither they had been taken by the Peruvians to dig guano; it spread from that centre with such intensity over the neighbouring islands that some valleys (Happar, Typee) were quite depopulated, while the total population lost, on an estimate, about one fourth of its number. Since that time vaccination has been introduced by the French authorities. On the other hand, some island groups of Polynesia appear still to enjoy an absolute immunity from smallpox; such as the *Tonga* and *Fiji* Archipelagoes, the *Samoa Islands*, which were quite exempt up to 1860 at least,[1] and the *Gambier* group, which Brassac gives as still exempt in 1876.[2]

On *European* soil, the smallpox up to the beginning of this century, or to the introduction of vaccination, had been one of the most widely distributed, most frequent, and most destructive of pestilences. In the southern countries, as we have seen, the disease had obtained a firm footing in the sixth century, and it appears to have been after that date that it penetrated to the northern regions. In the *Netherlands* the first account of it dates from the tenth century; the chronicles make mention of the death from smallpox of the Countess Elfrida in 907, and of Count Arnold of Flanders in 961,[3] the term "variola" occurring in the latter case. In *Denmark* it must have been already prevalent in the thirteenth century; for *Iceland*, which was first visited by smallpox in 1306, received the infection from Denmark. From that date down to recent times, it has been nineteen times epidemic in Iceland, undoubtedly reintroduced each time by ships, especially Danish. Since the introduction of vaccination it has only once, in 1819, attained a very wide diffusion; later outbreaks (1836, 1839, and 1840) have been prevented by strict isolation of the sick,[4] and Finsen, who writes under date 1874, says that he has not seen a single case of smallpox within the last ten years.[5] In the chronicles of

[1] Turner, 'Nineteen Years in Polynesia,' London, 1861, p. 536.

[2] 'Arch. de méd. nav.,' 1876, July, p. 12.

[3] 'Thijssen, 'Beschouwing der Ziekten in de Nederlanden,' Amsterdam, 1824.

[4] Schleisner, 'Island undersögt fra et laegevidenskabeligt synspunkt,' Kjöbenh., 1849, p. 50; Panum, in 'Verhandl. der Würzb. Phys.-med. Gesellschaft,' 1852, ii, p. 295.

[5] 'Jagttagelser angaaende sygdomsforholdene i Island,' Kjöbenh., 1874, p. 47

Ireland the occurrence of the disease is first mentioned in the fifteenth century, but no definite information about epidemics of smallpox there occurs until the eighteenth.[1] In the *Faröe Islands* it has been prevalent only twice, the first time in 1651,[2] and again in 1705; each time it was imported from Denmark and proved very destructive. Since the latter date this group of islands has been free from it.[3] Even at the present day smallpox takes no inconsiderable place in the aggregate sickness of European countries. But since the introduction of vaccination, and especially since its legal enactment and official supervision, the occurrence of the disease has been confined within tolerably narrow limits. Only in large and densely populated towns is the malady kept up continuously by successive importations and reproduction of the morbid poison. From time to time, as the number of susceptible individuals reaches a greater height, it breaks out in epidemics which often spread widely; but the disease in Europe has never, since the beginning of the century, attained that frightful importance which it had in past centuries. How great that importance was will appear in the sequel, in the comparative account of the mortality from smallpox in the several countries of Europe.

For the *Western Hemisphere* the history of smallpox begins soon after the landing of the first European immigrants. To whatever places they came at length and there settled, everywhere they carried the disease with them and gave it to the natives. But a still more terrible source for America was the importatation of negro slaves; so much so that in after years, particularly in South America and the West Indies, not only the first appearances of smallpox, but every fresh outbreak of it, could be traced to importation from Africa.[4]

The first outbreak of smallpox in the Western Hemisphere took place in the West Indies in 1507, fifteen years after the discovery of America, and it was so disastrous that whole tribes were extirminated by it. I have not succeeded in

[1] Wylde, in 'Edin. Med. and Surg. Journ.,' 1845, April, p. 250.

[2] Debes, in 'Bartholini acta med.,' Hafn, 1673, i, p. 86.

[3] Manicus, in 'Bibl. for Läger,' 1824, pt. i, p. 32; Panum, l. c.

[4] Chapman, 'Lectures on the more important Eruptive Fevers,' Philad., 1844; Chisholm, 'Essay on the Malignant Pestilential Fever, &c.,' London, 1801, i, p. 60; Desportes, 'Histoire des Maladies de S. Domingo,' Paris, 1770, i, p. 89.

finding out how long this epidemic lasted or how far it spread. The next information about the disease dates from 1517, in which year the sickness was imported by the Spaniards into Hayti. The subsequent outbreaks in the West Indies are chiefly connected, as has been said, with the importation of negro slaves. Thus, as late as 1819 it was introduced into Martinique by a slave-ship ;[1] and even the outbreak of 1851 in Jamaica was connected with the arrival of free negro labourers from the Gold Coast.[2] Generally speaking, the visitations of smallpox in the West Indies down to recent times have been severe ; thus, in the epidemic of 1843 in St. Thomas, at least one sixth of the population sickened,[3] and only those islands where vaccination has found somewhat general acceptance, such as Antigua[4] and Jamaica,[5] have enjoyed a comparative immunity. The disease reached *Mexico* for the first time in 1520 with troops from Spain ;[6] the number of persons swept off in a short time has been estimated at three millions and a half. There is mention of later epidemics of especial severity in the years 1763, 1779, and 1797. In 1804, vaccination was introduced into Mexico, but only in a small way ; so that the country down to the most recent times has often had to bear very severe smallpox epidemics.[7]

The *Eastern seaboard of the United States* was reached first in the beginning of the seventeenth century, the disease having shown itself in Boston in 1649 ;[8] here also vaccination, which was early introduced (1799), put a stop to the sickness in its disastrous forms, and inasmuch as the larger part of the interior (Mississippi Valley) was not colonised until after the introduction of vaccination, the dis-

[1] Account in 'Nouv. Journ. de Méd.,' 1819, May, p. 67.

[2] Miller, in 'Med. Times and Gaz.,' 1867, April, p. 441.

[3] Account in 'Sundhedskoll. Forhandl.' for 1844, p. 3.

[4] Nicholson, in 'Transact. of Epidemiol. Soc.,' 1866, iii, p. 48.

[5] Seaton, in 'Assoc. Med. Journ.,' 1855, p. 728.

[6] Bernardo Diaz, in his 'Hist. verdadera de la conquista de Nueva España' (Madrid, 1632), states as an eye-witness: "Y como veniuros en aquel tiempo con cortés, y dende á diez meses vino Narvaez, y truxo un negro lleno de virnelas; et qual las pegó á todos los Indios que habia en un pueblo, que ce decia Cempoala, é desde aguel pueblo cundió toda la Nueva-España, é ovo grande pestilencia."

[7] Compare Stricker, in 'Hamb. Zeitschr. f. Med.,' 1847, xxxiv, p. 525; Müller, 'Monatsblätter für Med. Statist.,' 1857, No. 6; Jourdanet, 'Le Mexique, &c.,' Paris, 1864, p. 406.

[8] Brown, in 'Amer. Med. Recorder,' 1829, Jan., p. 50.

case there, as in Europe subsequently to vaccination, never attained a wide diffusion among the whites.[1] On the other hand, smallpox made frightful havoc among the Indian tribes, following the march of colonisation westward. Thus in Kansas it broke out in a destructive form among the natives first in 1837,[2] and in California in 1850, after the arrival of the gold-diggers.[3] In the same way, it has in more recent times taken an enormous number of victims among the Indian population of British Columbia;[4] in Vancouver's Island, more than a thousand Indians died in the winter of 1862-63, and there was considerable anxiety lest the whole native population should be cut off.[5] It was introduced first to *Greenland* from Denmark in 1733; and its outbreak was so disastrous there that almost the whole colony died. That country has had repeated visitations of the sickness since then (1800, 1809, and 1851).[6]

Its first appearance, as well as its later outbreaks, on the continent of *South America*, are for the most part due to importation of the disease by negro slaves from Africa, and this applies particularly to Guiana and Brazil. In *Guiana*, smallpox has occurred but seldom, no doubt by reason of the extremely limited traffic in the country; thus Bajon, during a residence of many years in Cayenne, saw only one epidemic (1766), which owed its origin to an importation of negroes.[7] The malady broke out in 1803 under the same circumstances; Nogen says in his account of this epidemic:[8] "The alarm among the colonists was all the greater that this malady is not at all endemic in Guiana, and this was its first appearance." The disease first reached

[1] Drake, 'Treatise on the Principal Diseases of the Interior Valley of North America,' Philad., 1854, ii, p. 565.

[2] Lloyd, in the English transl. of Prince Maximilian's 'Reise im Innern v. Nord-America.'

[3] Praslow, 'Der Staat Californien in med.-geogr. Hinsicht,' Gött., 1857, p. 55.

[4] Freyman, in 'Arch. für wissenschaftl. Kunde Russlands,' 1848, vi, p. 226.

[5] Account in 'Philad. Med. News,' 1863, p. 32.

[6] Wendt, 'Efterretninger om Börnekoppor, &c.,' Kjöbenh, 1824, p. 67; Lange, 'Bemaerkn. om Grönlands sygdomsforhold,' Kjöbenh, 1863, p. 34.

[7] 'Nachrichten zur Geschichte von Cayenne.' From the French. Erfurt, 1780, ii, p. 56.

[8] 'Revue méd.,' 1834, Aug., p. 313.

Brazil, according to Piso,[1] in 1560, and was in like manner introduced by negroes; it has been very prevalent there, particularly among the Indians, down to recent times, in spite of vaccination, which was introduced in 1814 but has been carried out unquestionably in a very careless and imperfect way; so disastrous has the smallpox been that Tschudi speaks of it as the chief scourge of the country.[2] Later epidemics as well, such as that of 1834, have always followed the arrival of slaveships.[3] Smallpox was probably brought to the *States of the La Plata* by the Spaniards, but there is nothing accurately known of the time of its first occurrence there;[4] down to recent times it has over and over again committed great ravages throughout the country. The same is true of *Chili,* where the disease had been introduced previous to 1554,[5] also by the Spaniards; all the chroniclers agree in describing it as the most frightful scourge of the inhabitants,[6] and on that account, as Fournier states,[7] it is known to the people as "peste" κατ' ἐξοχήν. In *Peru* (in Lima particularly) the first epidemic of smallpox is said to have taken place in 1802 (?); it is now one of the greatest plagues of the country, particularly among the negroes and Indians,[8] notwithstanding that the natives appear to have been acquainted with the protective power of vaccine long before the advent of Jenner.[9]

[1] 'De utriusque Indiæ historia naturali et medica,' Amsterd., 1658.

[2] 'Wien. med. Wochenschr.,' 1858, No. 31. An account of the destructive ravages of smallpox in the province of Maranhão is given by Plagge in 'Monatsbl. für Statist.,' 1857, No. 10.

[3] Sigaud, 'Du climat et des maladies du Brésil,' Paris, 1844, 108, 181.

[4] Brunel, 'Observ. topogr. et méd. faites dans le Rio-de-la-Plata,' Paris, 1842, p. 42.

[5] Coni, in 'Virchow-Hirsch's Jahresbericht,' 1878, ii, p. 46.

[6] Pöppig, in 'Clarus' Beiträge zur Heilkd.,' 1834, i, p. 526; account in 'Arch. de méd. nav.,' 1864, Sept., p. 103; Boyd, in 'Edin. Med. Journ.,' 1876, Aug., p. 110.

[7] 'Arch. de méd. nav.,' 1874, Sept., p. 148.

[8] Smith in 'Edin. Med. and Surg. Journ.,' 1840, April, p. 333.

[9] Unanue ('Observ. sobre el clima de Lima, &c.,' Lima, 1806) mentions the following fact of interest in this connexion:—A negro slave having been vaccinated on the outbreak of the disease in 1802, but without effect, stated, when the operation was about to be repeated on him, that he was sure he could never take the smallpox as he had got an eruption at the cow-milking in the Andes, which, the shepherds had told him, had come from contact with a nodular eruption on the cows' udders, and would act as a protection against small-

§ 36. Influence of Vaccination on the Prevalence of Smallpox.

However rich the medical and historical literature may be in accounts of the occurrence of smallpox at various parts of the globe; however clearly the past may bear witness to the amount of human life that has been offered up a sacrifice to this murderous disease, beside which the loss through the bloodiest of wars, or through other severe pestilences such as plague and cholera, appears to be infinitesimally small; and however sufficient may be the researches into the present geographical distribution of the disease,—still the materials available to us are not even in a measure adequate for drawing up a proper history of the pestilence in past centuries. It was not its rarity but, as Häser justly remarks, actually its every-day occurrence that weakened the interest of epidemiographists towards it. But we may be all the more easily consoled for this defective state of the epidemiological record of smallpox in the pre-vaccination era, by the reflection that even a complete narrative of the smallpox epidemics of that time would have no further scientific interest than as enabling us to vindicate the importance of vaccination for preventing the disease, or, at least, for reducing the mortality caused by it. So far as answering *that* question goes, the materials at our service are amply sufficient; and the question has been already answered so often and so thoroughly, particularly in the classical reports of the English Board of Health under the editorship of John Simon,[1] and in the excellent work of Bohn,[2] that I find no occasion to adduce once more the historical proofs relating to it.

That the achievement of Jenner was at once a turning-point in the history of smallpox, and a new era in the physical welfare of mankind; that the power of the pestilence became more and more restricted both in range and in severity in proportion as the practice taught by him

pox. From this statement it follows that the disease had been prevalent in Peru (at least in the Andes) long before 1802.

[1] 'Papers relating to the History and Practice of Vaccination,' London, 1857.

[2] 'Handbuch der Vaccination,' Leipzig, 1875.

obtained acceptance and careful attention at the hands of various nationalities; that the disease at the present day, as is abundantly shown in the foregoing account of its geographical distribution, still bears, in those regions where ignorance and prejudice have opposed the adoption of vaccination or where the carelessness of the authorities has neutralised its good effects, the same character for destructiveness that meets us in the medical and chronological accounts, and in the mortality statistics, of European countries in the pre-vaccination period; that even to-day we find in the devastation of populous districts, and in the uprooting of whole tribes, the indications of what this ravaging pestilence could do—all this is so thoroughly brought out in the writings I have named, that it can be only folly or stupidity that would seek nowadays to minimise or to question the immortal merits of Jenner. The foolish attempts made to discredit vaccination may be met with the simple but conclusive remark of Porter's :[1] "it will require an immense accumulation of facts, more than the world ever saw, to shake our faith in the protective influence of vaccination."

The expectations, indeed, which arose out of Jenner's original practice for warding off the smallpox have not been borne out to the full extent. Experience has taught us that the practice required to be widened, that the protective power of vaccine was found in many cases to be sufficient only for a certain time, that the susceptibility to the morbid poison, abrogated by a single vaccination, was in many persons restored after a longer or shorter period, and that re-vaccination was necessary to give lasting protection. Experience also has served to prove that the supervision of vaccination by the State, even in those countries where it was carried out according to law, had not been, and even still in part is not, administered with the energy that can alone make the guarantee perfect.[2] It has become clear that this guarantee

[1] 'Amer. Journ. of Med. Sc.,' 1853, Oct., p. 322.

[2] Not to instance the futile efforts of European governments to introduce vaccination into their foreign possessions or colonies, many countries in Europe furnish the most telling evidence that vaccination and revaccination are still imperfectly carried out. Thus, to quote only a few examples, it is stated by Kanzow ('Sanitätsbericht aus dem Regbz. Potsdam von den Jahren 1869—1874,' Potsdam, 1876, p. 23), in treating of the smallpox epidemic of that

can be secured only where the indifference and prejudice of the public, often led astray by false prophets, is met by compulsory vaccination, and only where this important matter is not left to police edicts or to administrative orders, but is regulated by statute. The Bavarian Government, not to mention others, has given a striking example of what may be achieved in this way.

With the introduction of vaccination into the civilised States of Europe, covering a period from 1799 to 1804, a remarkable decrease in the amount of smallpox and in the mortality caused by it, quickly became noticeable;[1] and thus

district in 1871-72, that the considerable extension of the disease (the mortality having been at the rate of 2·7 per 1000 of the population) had been much helped by the circumstance that the number of persons remaining unvaccinated had increased very much; and, as several physicians unhesitatingly testified, those only died of the disease who had never been vaccinated or had been vaccinated badly and without effect. The condition of things was no better in the other departments of Prussia and in the other States of Germany. Grimshaw ('Dubl. Journ. of Med. Sc.,' 1878, Jan., p. 490) remarks of the Dublin epidemic of 1871 that great laxity in carrying out vaccination had occurred there in recent years, and that a wide field for the ravages of the disease had in that way been left open. The complaints from France of the neglect of vaccination of late have been especially strong. Vacher ('Gaz. méd. de Paris,' 1875, No. 38, p. 471), in his report on the smallpox epidemic of 1870-71 in France, writes:—"It is almost incredible that, seventy-five years after Jenner's discovery, one third of the French people should be without the benefit of vaccination; there are departments, such as L'Aveyron and La Corse, where one can count hardly more than twenty vaccinated among one hundred of the inhabitants." Guillon, who served in the Franco-Prussian War of 1870-71 as surgeon to a batallion of Gardes Mobiles 1158 strong, and had under observation 600 cases of smallpox among them, complains in like manner of the extremely defective vaccination and revaccination in France, and appends to his report ('Sur une épidémie de variole,' Paris, 1871, p. 37) the following note:—"During his enforced stay with the Germans, my excellent friend Dr. Jules Petit remarked the almost complete immunity which our enemies enjoyed as regards the epidemic of variola. On his inquiring of the Prussian surgeons the cause of this, they told him that the result was exclusively owing to compulsory revaccination." I add to this the interesting fact given by Macpherson ('Indian Annals of Med. Sc.,' 1852, Jan., p. 232) that, in the Presidency of Bengal, at a time when smallpox was making frightful ravages among the natives, only 103 cases (with 29 deaths) occurred among 84,143 European troops in a space of four years, 11 cases (with 1 death) among 2970 officers, 26 cases (with 4 deaths) among 7941 soldiers' wives, and 26 cases (with 9 deaths) among 9255 soldiers' children.

[1] It has been proved conclusively in the writings above mentioned (the report published by the English Medical Department, and the work of Bohn, p. 289 ff), that the introduction of vaccination was not merely an accidental coincidence with one of those temporary remissions of the disease which had been often observed

it came to be believed that the enemy had been driven for ever from the field. The peace had lasted, however, only some ten or fifteen years, when the ravaging disease raised its head anew. And if its prevalence on European and North American soil, as well as in all those regions where vaccination had found general acceptance, was no longer to the extent, and above all of the malignancy, of previous centuries, yet there were many epidemics, more or less widely spread, and sometimes covering a great part of the globe, which vividly recalled the tragedies of the past. In the post-vaccination epoch, the disease has been most severe and of a truly pandemic character during the years from 1868 to 1873.[1]

in previous centuries. I may here remark further that in the history of pestilences in Italy by Corradi ('Annali delle epidemie occorse in Italia,' Bologna) there is not a single epidemic of this disease mentioned for the years from 1805 to 1817, contrasting with the numerous smallpox epidemics of the 18th century. The following table, drawn up by Seaton for the English reports, of the mortality from smallpox in the various countries of Europe in the pre-vaccination and post-vaccination periods, affords very interesting evidence on this point.

Period		Locality.	Mean annual mortality from smallpox per million population	
Before vaccination.	After vaccination.		Before vaccination.	After vaccination.
1777—1806	1807—1850	Lower Austria ...	2,484	340
1777—1806	1807—1850	Upper Austria and Salzburg	1,421	501
...	...	Styria	1,052	446
...	...	Illyria	518	244
1777—1806	1838—1850	Trieste	14,046	182
1777—1806	1807—1850	Tyrol	911	170
...	...	Bohemia	2,174	215
...	...	Moravia	5,402	255
...	...	Austrian Silesia ...	5,812	198
...	...	Galizia	1,194	676
...	...	Bukowina ...	3,527	516
1776—1780	1810—1850	East Prussia ...	3,321	556
1780	1816—1850	Posen	1,911	743
1776—1780	1810—1850	Brandenburg ...	2,181	181
1776—1780	1816—1850	Westphalia ...	2,643	114
...	...	Rhine Province ...	908	90
1781—1805	1810—1850	Berlin	3,442	176
1780	1810—1850	Pomerania ...	1,774	130
1774—1801	1810—1850	Sweden	2,050	158
1751—1800	1801—1850	Copenhagen ...	3,128	286

[1] A tolerably complete survey of the smallpox epidemics of the last fifty years is given by Bohn (l. c., pp. 19 ff). Seaton has given ('Report of the Med.

§ 37. Periodicity of Epidemics.

Not many of the acute infective diseases show in their incidence and diffusion so complete an independence of the *conditions of climate and soil* as smallpox, which thrives equally well wherever its contagion is carried, and wherever it finds a population open to its reception and capable of reproducing it. It is this last circumstance that explains in the most obvious way the seeming *periodicity in the succession of the epidemics* at various points, a periodicity which had engaged the active attention of the earlier observers both before and after vaccination, and has given rise to many metaphysical explanations, including the theory of a "constitutio epidemica variolosa" periodically developing itself. The first point that strikes one particularly, in a criticism of these observations, is that this periodicity in the recurrence of smallpox epidemics has presented itself in very various ways at the various places of observation. Thus, Werlhof,[1] after forty years' experience in Hanover, estimates the interval at four to five years, and it is put at the same figure by Heineken[2] for Bremen, and by Gibson[3] for the Deccan; while Guys assigns three to four years for Aleppo,[4] Hufeland five to six years,[5] Holwell, Heymann,[6] Dawson,[7] Bayfield,[8] and others seven to eight years for India, and Stricker sixteen years for Mexico.[9] All these data are based upon observations for very short periods; the circumstances will look entirely different when we take account of the epidemic outbreaks of the disease at a given place over a longer period and in the times before vaccination as well as after it. I content

Officer of the Privy Council, &c., for the Year 1874,' new series, iv, Lond., 1875, p. 51) a detailed account of the great epidemic of 1869-73.

[1] 'Disquis. de variolis' in Opp., Hanover, 1775, p. 477.

[2] 'Die Hansestadt Bremen, &c.,' Bremen, 1836.

[3] 'Bombay Med. Transact.,' ii, p. 10.

[4] 'Statistique du Paschalik d'Alep.,' Marseille, 1853, p. 63.

[5] 'Bemerk. über die Blattern, &c.,' Leipzig, 1789.

[6] 'Versuch einer Darstellung der Krankh. in den Tropenländern,' Würzb., 1855, p. 224.

[7] 'Philad. Med. Examiner,' 1852, May.

[8] 'India Journ. of Med. Sc.,' 1834, i, p. 362.

[9] 'Hamb. Zeitschr. für die ges. Med.,' 1847, xxxiv, p. 525.

myself with adducing a few examples from the history which are especially instructive in this respect. Epidemic outbreaks of smallpox occurred as follow:

At Boston,[1] 1649, 1666, 1678, 1690, 1702, 1721, 1730, 1752, 1764, 1776, 1788, 1792; at Philadelphia,[2] 1808, 1811, 1823, 1827, 1833, 1841, 1845, 1848, 1851, 1855, 1860; at Nancy,[3] 1825, 1832, 1841, 1846, 1850; at Vienna,[4] 1742, 1745, 1749, 1757, 1759; at Breslau,[5] 1804, 1813, 1823, 1831, 1842, 1851, 1856, 1863, 1868, 1871. Réunion[6] was quite free from smallpox for twenty-four years (1827-1851) and the epidemic of 1851-52 was followed seven years later by that of 1858-60. In Iceland the disease was prevalent in 1306, 1310, 1347, 1380, 1430, 1511, 1555, 1574, 1580, 1590, 1616, 1632, 1636, 1655, 1658, 1671, 1707, 1742, 1762, 1786, and so on.

There are, in my opinion, two factors only that determine the recurrence of an epidemic of smallpox: on the one hand, the necessary number of persons susceptible of the morbid poison, and, on the other hand, the introduction of the virus itself. In large towns where there is certainly no lack of frequent importations of the kind, where, in fact, the poison is perennial, an epidemic will come about, as Fleischmann assumes for Vienna,[7] whenever there is a sufficiently large number of persons capable of infection, that is to say, in our post-vaccination times, unvaccinated; and therefore four or five years may be regarded as sufficient for Vienna. But there can be no rule of the kind whenever the practice of vaccination is carried out at all efficiently. Förster,[8] who forms his estimate in the same way as Fleischmann, puts the frequency of epidemic recurrences at Dresden at seven to eight years; but, for those places where the morbid poison dies out at the end of an epidemic, it stands to reason that the disease cannot reappear until new virus is imported, and under these circumstances there can be absolutely no question of regularity in the

[1] Curtis, in 'Transact. of the Amer. Med. Assoc.,' 1851, ii, p. 487.
[2] Jewell, in 'Amer. Journ. of Med. Sc.,' 1862, April, p. 378.
[3] Simonin, 'Recherch. topogr. et méd. sur Nancy,' Nancy, 1854, p. 244.
[4] Plenciz, 'Tract. de variolis,' Vienna, 1762, p. 49.
[5] Pastau, in 'Arch. für klin. Med.,' 1873, xii, p. 112.
[6] Azéma, l. c.
[7] 'Jahrb. für Kinderheilkunde,' 1870, iii, p. 456.
[8] Ib., 1868, i, p. 121.

recurrence of the sickness. This is naturally best seen at those places which are remote from traffic and much more rarely infected than places lying in the track of commerce. Popper[1] for Prague, and Hagenbach[2] for Basel, completely dismiss the idea of regularity in the recurrence of smallpox epidemics, and they are perfectly justified in doing so, upon their data.

§ 38. Influence of Season.

Although the occurrence of smallpox is seen to be in general independent of conditions of climate, still the *season of the year* has a marked influence upon the amount of the sickness or upon its epidemic diffusion. Razes[3] had already arrived at the conviction, from his experiences in Arabia, that epidemics of smallpox occur at all seasons, but that they mostly begin towards the end of autumn and in the early spring, or in the cold season. Opinions completely agreeing therewith have been expressed by Pruner,[4] who adds that "the hot months in the latitude of Cairo, ordinarily from June to October, are for the most part equally little suited for the development of smallpox as for the plague;" and by Rigler[5] for Constantinople, where smallpox appears usually on the approach of winter, continues till spring, and dies out in the hot season; by Ferrini for Tunis, where the disease prevails most frequently in spring; by Rendu[6] for Brazil, where the time for the prevalence of the disease falls in the months from October to December (that is, in spring); by Pöppig[7] for Chili, where the epidemic is at its height in the months from July to November (end of winter and spring); and by Pearson,[8] Morache,[9] and Lagarde[10] for China.

[1] 'Zeitschrift für Epidemiol.,' 1876, ii, p. 222.

[2] 'Jahrb. für Kinderhlkd.,' 1875, ix, p. 62.

[3] 'Liber de variolis,' cap. ii, after the translation by Mead. Opp. Naples, 1752, p. 37.

[4] L. c., p. 128.

[5] L. c., ii, p. 30.

[6] 'Etud. topogr. et méd. sur le Brésil,' Paris, 1848, p. 66.

[7] L. c., p. 526.

[8] L. c., p. 361.

[9] L. c.

[10] L. c.

The following table gives the details as to the amount of sickness in India in the various seasons:

Deaths from Smallpox.

Place.	Years.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	January.
Calcutta[1]	1832–50	2372	3689	2846	1419	761	551	189	181	134	120	512	1316
Bombay[2]	1846	11·17	20·34	24·24	17·47	11·36	4·51	2·20	1·21	0·51	0·90	1·84	4·18
Bombay[3]	1865	368			111			21			66		

The greatest prevalence, accordingly, falls here in the cold season corresponding to the spring of temperate latitudes, and that applies to the whole of Bengal,[4] the Madras Presidency,[5] the districts belonging to the North-West Provinces,[6] and to the Punjaub.[7]

The frequency of the disease in the several seasons within temperate latitudes shows an almost complete correspondence with the above. The following table gives the time when the epidemic was at its height in ninety-nine outbreaks on European or North-American soil, of which there are accurate data.

October to January... 3		
October to March ... 2	Autumn and Winter	10
November to December 2		
November to January 3		
December to January 3		
December to March... 4	Winter	17
January to March ... 8		
February to March... 2		

[1] 'Report of the Smallpox Commissioner,' Calcutta, 1850.

[2] Morehead, 'Clinical Researches on Diseases in India,' Lond., 1856, i, p. 317. The numbers are the monthly proportion in each hundred fatal cases.

[3] Macpherson, in 'Med. Times and Gaz.,' 1873, July, p. 31.

[4] Maclean, in 'Calcutta Med. Transact.,' v, p. 399.

[5] Macpherson, Cornish, ll. cc.; Day, in 'Madras Quart. Journ.,' 1861, Oct., p. 213.

[6] McGregor, Macpherson, ll. cc.

[7] De Renzy, in 'Brit. Med. Journ.,' 1871, Sept., p. 264.

December to May	4			Cold Season 67
January to April	7			
January to June	1	Winter and Spring	16	
February to April	3			
February to June	1			
March to April	4			
March to May	9	Spring	24	
March to June	7			
April to May	4			
April to September	2			
May to June	2	Spring and Summer	7	
May to August	3			
June to July	6			
June to August	7	Summer	14	Warm Season 32
August to September	1			
August to October	6	Summer and Autumn	6	
September to October	2			
October to November	3	Autumn	5	

This result is borne out by the following table, in which I have put together the number of deaths from smallpox within a certain time, in various countries or places in the temperate zone, according to the months or quarters of the year.

		Winter.			Spring.			Summer.			Autumn.		
		January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.
Danzig	1871–72	368	351	354	454	493	461	195	138	116	126	136	135
Breslau	1863–64	199	144	69	52	22	27	24	36	58	69	128	147
Berlin	1863–67	96	125	129	154	200	192	141	78	75	91	82	94
Netherlands	1870–73	2005	2501	2987	2966	2531	2064	1361	939	697	637	715	1172
Paris	1819–26	336	224	156	142	178	221	403	669	832	983	639	415
Paris	1860–69	569	517	488	425	364	292	298	289	342	418	506	701
Paris	1870	174	293	406	561	786	914	1072	713	700	1361	1722	1837
Milan	1870–71	149	83	90	179	328	388	451	643	829	953	961	590
Philadelphia	1871–72	832	615	536	260	149	122	35	18	18	236	502	1104
		4728	4853	5215	5193	5051	4681	3980	3523	3667	4874	5391	6195
			14,796			14,925			11,170			16,460	
England	1837–40		9,095			9,470			10,005			12,483	
England	1851–52		389			472			221			413	
England	1871–72		12,623			13,533			7,565			8,045	
Sweden	1856–62		2,030			1,753			783			691	

On the other hand, the character or severity of the disease appears to be quite uninfluenced by the season and the kind of weather. Sydenham, indeed, thought himself justified in

concluding from his observations, made in the London epidemics of 1667-69 and 1673-75, that smallpox ran an especially severe course in the high temperature of summer, and Stoll[1] afterwards expressed the same opinion. However, as Van Swieten was the first to remark,[2] there are numerous exceptions to this rule, and his presumption that the severity of the epidemic depended on other things than the influences of the season, has been borne out by subsequent experiences, such as those of the very severe winter epidemics of 1666 in Paris,[3] 1840 in Semécourt,[4] 1847[5] and 1869-70 in Paris,[6] 1726 in York,[7] 1761 in Göttingen,[8] 1754 in Lüneburg,[9] 1798 in Cracow,[10] 1801 in Treves,[11] and of 1806 in Helmstädt.[12] Pearson, in fact, states[13] that in China the disease is the more severe the earlier in the year the epidemic breaks out, or the lower the temperature at the time of the outbreak.

§ 39. The Specific Poison of Smallpox.

That smallpox owes its origin to a *specific* poison, that this poison is capable of reproduction, and is therefore an organic body, and that it can at once exert its power in the form in which it is eliminated from the sick person, wherein it bears the clear mark of a contagious morbid poison—all this hardly requires to be proved, even although the investigations[14] that have been directed to the detection of the

[1] 'Ratio medendi in nosoc.,' Vindob., ii, 211.

[2] 'Comment. in Boerhaave Aphorismos,' Lugd. Bat., 1772, v, 5.

[3] Lamotte, 'Traité complet de chirurgie,' Paris, 1722, iii, p. 383.

[4] Bastien, in 'Travaux de la Soc. des Sc. méd. du départem. de la Moselle,' 1841-43, Metz, 1843, p. 2.

[5] Matice, in 'Gaz. méd. de Paris,' 1847, Oct., p. 797.

[6] Besnier, in 'Union méd.,' 1870.

[7] Wintringham, 'Comment. nosol.,' Berl., 1791, p. 85.

[8] Hensler, 'Observ. de morb. variol.,' Gött., 1762.

[9] Lentin, 'Beiträge zur Arzneiw.,' Leipzig, 1797, i, p. 223.

[10] Account in 'Med. Nationalztg.,' 1798, p. 666.

[11] Burchardt, in 'Med. Annalen,' 1802, Correspondbl., 177.

[12] Remer, in 'Hufeland's Journ.,' 1815, xl, pt. 4, p. 32.

[13] 'Calcutta Med. Transact.,' vi, l. c.

[14] Keber, in 'Virchow's Archiv,' 1864, vol. 42, p. 112; Chauveau, in 'Gaz. méd. de Paris,' 1868, p. 140; Salisbury, 'Microscopic Examinations of Blood and Vegetations found in Variola,' &c., New York, 1868; Weigert, in 'Centralbl.

poison have either had very doubtful results or no result at all.

As to the *native habitat of the smallpox poison* there is still, as we have seen at the commencement of this inquiry, much obscurity. The assumption that it is coextensive with the distribution of the disease is contradicted by the evidence adduced above, that large portions of the globe, the whole Western Hemisphere, the Continent of Australia, and Polynesia, have enjoyed immunity from smallpox until such time as the disease was imported thither from other points, and that many of these regions had not lost that immunity until quite recent times. From its native foci, the morbid poison has been spread gradually, and doubtless repeatedly, by sick persons or by objects to which it clung, over the greater part of the globe, and thereby the disease has been diffused in ever-widening circles. Contrasting, however, with other morbid poisons, such as those of cholera, typhus, and yellow fever, that retain the power of reproduction for only a short time away from their native focus, the poison of smallpox appears able to survive outside its habitat so long as it finds individuals who are susceptible to it, or who offer to the poison a suitable soil for its reproduction. It is when that nutrient soil fails that the virus perishes, or loses its potency and capacity for increase. Evidence of this is furnished by the behaviour of the disease, as above described, in regions such as Iceland, the Faröe Islands, Guiana, and the like, which are situated remote from the great lines of traffic, or such as Réunion, the West Indies, and the Australian continent, which are connected with the centres of commerce by the sea-road only; in those regions the introduction of the morbid poison takes place much more rarely than by the quicker way of land traffic, and there are accordingly decades intervening between the various epidemic outbreaks of smallpox, during which the country enjoys a complete exemption from the disease. It is otherwise in the great centres of intercourse, where the morbid poison is continuously provided with a suitable

der med. Wiss.,' 1871, No. 391, 'Anat. Beiträge zür Lehre von den Pocken,' 2 pts., Breslau, 1874 and 1875, and in 'Deutsche Zeitschr. für pract. Med.,' 1874, No. 43; Zülzer, in 'Berl. klin. Woch.,' 1872, No. 51; Cohn, in 'Virchow's Archiv,' 1872, vol. 55, p. 229.

"nutrient soil" in the fluctuating population, where the disease therefore continues to flourish, and will attain epidemic diffusion whenever predisposed persons are massed together more than usual, and more particularly when there are active movements amongst the population. In fact, these are just the centres from which all the far-reaching epidemics take their start.

§ 40. Individual Susceptibility to the Poison.

The single factor, then, that determines the development of the sickness, besides the presence of the infecting substance, is a population susceptible to it; and this *susceptibility to the poison of smallpox* is one that extends to the whole of mankind. Experience shows, however, that the coloured races, and especially the *negro race*, are, *ceteris paribus*, in greater risk from smallpox than the whites.

"The members of the human family," says Pruner,[1] "that are most susceptible to the poison of smallpox are the negroes. Not only in their native lands, but in other parts of the world as well, they are the first to succumb to the epidemic influence, and also the last. It is no unusual thing to see negroes attacked by smallpox as soon as they arrive in Egypt (where they certainly change their way of living as well as the climate) and that, too, at times when the disease does not exist among the other inhabitants." Similar statements, equally emphatic as to the increased intensity of the disease in the negro, are made by Daniell[2] for the West Coast of Africa, for Martinique by Rufz,[3] (according to his observations in the epidemics of 1836-37 and 1848-50) for Curaçao,[4] for Cayenne[5] by Bajon, and for Peru.[6] In Boston there died in the epidemics of 1649-1792, or at a time when there was no question of protection by vaccination, 10·8 per cent. of white patients and 23·7 per cent. of black.[7]

[1] L. c., p. 120.
[2] L. c., p. 41.
[3] 'Arch. de méd. nav.,' 1869, Aug., p. 137.
[4] Account in 'Nederl. Tijdschr. voor Geneesk,' 1862, vi, p. 592.
[5] L. c., p. 56.
[6] 'Arch. de méd. nav.,' 1864, Sept., p. 188.
Shattock, in 'Amer. Journ. of Med. Sc.,' 1841, April, p. 372.

In the epidemic of 1850 at Baltimore, the proportion of deaths among 10,000 white inhabitants was 8·1, and among negroes 14·5.[1]

§ 41. Diffusion of the Morbid Poison.

The *dispersion of the morbid poison* takes place either by the smallpox patients themselves, or through the medium of other persons, or of articles to which it clings. It has been conclusively proved by very numerous and unambiguous observations that an atmosphere of smallpox poison develops around the sick, especially when they are crowded in close rooms; or, in other words, that the air may become a carrier of the contagion, so that the latter can be spread by the atmospheric currents within a small range. There is certainly no mathematical expression to be found for the extent of that range; at the utmost, it extends no farther than the immediate surroundings of the sick, and the *smallpox wave* of Cornish,[2] which follows the direction of the east wind and determines the progress of the disease in India from east to west, is to be considered an idle fancy, all the more so that no such constant procession of smallpox has been observed in India or elsewhere.

§ 42. Relation to Chicken-pox.

As it required the experience of several centuries before the specific nature of smallpox was recognised by physicians, it is not surprising that a still longer time elapsed before we learned to distinguish between variola and *varicella*. There are, indeed, indications in Razes,[3] Avicenna,[4] and others of the early chroniclers of smallpox, that they had seen varicella as well as variola; and we find more precise statements on the latter disease in Ingrassias,[5] Guidi,[6] and Rive-

[1] Frick, ib., 1855, Oct., p. 326.
[2] 'Lancet,' 1871, May, p. 703.
[3] L. c., cap. 70.
[4] 'Canon,' lib. iv, fen. i, tract. iv, cap. 6, ed Venet., 1564, ii, 71,
[5] 'De tumoribus praeter naturam tract.,' Neapoli, 1533, p. 194.
[6] 'Ars univ. med.,' Venet., 1596, ii, lib. 13, cap. 6.

rius[1] of the sixteenth century, and in Sennert,[2] Diemerbroek,[3] and others of the seventeenth. But the merit of recognising and describing the characteristic features of varicella belongs to Heberden,[4] whose statements about the "chicken-pox" in England were followed by accounts of the same in France by Hatté;[5] while in Germany valuable facts about varicella and its relation to variola were furnished more especially by Heim.[6] The question of that relationship, whether it be one of identity or of specific difference between the two forms of disease, lies outside the limits of my task. In favour of the latter view, there are many data before us bringing out the fact that the geographical distribution of varicella as an endemic disease extends much farther than that of variola, that it was known in several parts of the world before the introduction of smallpox—at the Cape, in the southern territories of South America, and on the Australian continent—and that in after times it has continued, not unfrequently in epidemic form, to retain the distinctively mild character of its symptoms, altogether irrespective of importations of smallpox from time to time at long intervals, or of the introduction of vaccination.

[1] 'Method. curand. febr.,' sect. iii, cap. iii. 'Haq. Conist.,' 1651, p. 154.
[2] 'De febribus,' lib. iv, cap. 12.
[3] 'De variolis et morbillis,' cap. ii.
[4] 'Med. Trans. of the College of Physicians,' i, Lond., 1767, p. 427, and 'Comment. de morb. hist.,' cap. 69, ed. Lips., 1831, p. 229.
[5] 'La vérolette ou petite vérole volante,' Paris, 1759.
[6] Horn's 'Arch. für med. Erfahr,' 1809, x, p. 183.

CHAPTER V.

MEASLES.

§ 43. Old Views of "Morbilli:" the History Defective.

In the writings of the Arabian physicians,[1] to whom we owe the first scientific account of smallpox, reference is at the same time made to still another form of acute exanthem under the name of "hasbah," which was regarded by these observers as a modification of smallpox.[2] The same disease, always in association with smallpox, is mentioned also by the physicians of the middle ages under the various designations of morbilli,[3] rubeola,[4] rossalia, rossania, rosagia,[5] and the like, as well as under the colloquial names of "fersa" or "sofersa"[6] (Milanese), "mesles,"[7] afterwards "measles" (English), corresponding to the German "maal," and "masern" (German), the Sanscrit masura ("spots"). The few casual references to this disease in the Arabian physi-

[1] Razes, in 'Liber de variolis et morbillis,' also in 'Continens,' lib. xviii, cap. viii, Brix., 1486, fol. Bl. viii (besides the morbilli, there is mention also of exanthems under the names of "blacciae" and "lenticula"); further, in 'De re medica,' lib. x, cap. xviii (Opp. minor, Basil, 1544, p. 304), and in lib. division., cap. clix (Opp. e. c. 444); Ali Abbas, 'Liber theor.,' sermo viii, cap. 14, and 'Liber pract.,' sermo iii, cap. 1 (here the disease is named rubeola); Avicenna, 'Canon,' lib. iv, fen. i, tract. iv, cap. 8; Avenzoar, lib. ii, tract. vii, cap. ii.

[2] Thus it is stated in Avicenna: "Scias quod morbillus omnis est variola cholerica, et non est differentia inter ea ambo in plurimo reliquarum dispositionum, nisi quod morbillus est cholericus."

[3] Diminutive of morbus.

[4] Occurring only in the translation of the work of Ali Abbas (see previous note).

[5] In Concorregio, 'Practica de variolis et morbillis,' in 'Summula de curis febrium,' Venet., 1521, fol. 938.

[6] In Michael Scotus, 'De procreatione et hominis phisionomia,' cap. x; compare Gruner, 'De variolis et morbillis fragmenta,' Jena, 1790, p. 33.

[7] Joh. Anglicus, 'Praxis med,' Aug. Vindel, 1595, p. 1041.

cians do not go far towards characterising it, or towards deciding for us to what known forms of disease the "morbilli" correspond. In like manner, the Arabistic writings, which closely follow the Arabian writers, omissions and all, afford us no satisfactory information as to what we are to understand under "morbilli." Only now and then are individual symptoms mentioned, such as redness, flow of tears, throat affection, and the like, while the exanthem itself is described as spotted, papular, or vesicular. It is probable that we have here to deal with various exanthematous diseases; chiefly with scarlet fever and measles, but more particularly with scarlet fever, as we may infer from the statement made by several observers that the "morbilli" were as dangerous as smallpox, and often even more dangerous.

In the sixteenth century the views of physicians upon the acute exanthematous diseases had cleared up, so far, at least, as to recognise in the disease called "morbilli," or "rosalia," a morbid process different from that of smallpox. But under "morbilli" they still comprised measles and scarlet fever as being one and the same process,[1] and we meet with this obscurity as late as the seventeenth century, even after Sennert, Döring, Sydenham, and others had rightly discerned and taught the peculiarities of scarlet fever (first called "febris scarlatina" by Sydenham); so that even Morton, not to mention others, remarks in speaking of this disease:[2] "Hunc morbum prorsus eundem esse cum morbillis censeo," and adds: "exulet igitur per me e censu morborum haecce febris, nisi cuiquam 'morbillorum confluentium' titulo eum designare in posterum visum fuerit."

From the facts here stated we may conclude so much at least as to the *history of measles* in past centuries, that the disease was in all probability widely diffused over Asiatic and European soil during the middle ages; and it has retained that position as a sickness of the people in the centuries following. In the eighteenth century, by which time physi-

[1] A tolerably clear description of the rash of measles is given by the Roman physician, Prosper Marziani in his commentary on the 'Libri Epidemior.' of Hippocrates, Venet., 1652, p. 744. See Corradi, 'Annali delle epid. occ. in Italia,' parte iii, Bologna, 1870, p. 53.

[2] 'Pyretologia,' cap. v, Genev., 1696, p. 28.

cians had learned to recognise the peculiarities of the disease and to distinguish it from other forms of exanthem, we find many reports of more or less extensive epidemics of measles at numerous points in the Eastern and Western Hemispheres. At the present day the area of its distribution may be taken to extend over the whole habitable globe. The question of the *native seat of measles* baffles all research; the history teaches us, as will appear in the sequel, this much at least, that the sickness has not had a fresh origin at every point where it has occurred, that it has issued, and probably still issues, from certain centres which cannot be specified more particularly, and that the wider diffusion of the disease from these native foci, into regions nearer or more remote, depends upon a transporting of the morbid poison, which, like that of smallpox, can survive outside its habitat so long as there are found susceptible individuals, affording to the poison a soil adapted for its reproduction, while it perishes if there be no ground on which to reproduce itself.

§ 44. Present Geographical Distribution.

In Europe, both continental and insular, the distribution of measles reaches from the shores of the Mediterranean to the regions of the extreme north, which, if seldom visited, have still not been altogether exempt. In the *Faröe Islands*[1] the disease has been prevalent four times up to the present, first in 1781, then in 1846, again in 1862, but only to a slight extent, and lastly in 1875; it is proved to have been on every occasion imported, the last time from Shetland. Four epidemics have been observed hitherto in *Iceland*,[2] in 1664, 1694, 1846, and 1868;[3] these have originated in the same way as in Faröe, and so also have the numerous outbreaks of the sickness in *Lapland* (the last of them in 1852[4]). In the

[1] Schleisner, 'Island,' p. 51; Hjaltelin, in 'Dobell's Reports,' Lond., 1870, p. 283.

[2] Manicus, 'Bibl. for Läger,' 1824, i, p. 32; Panum, ib., 1847, i, p. 319, and in 'Virchow's Archiv,' i, p. 492; Madsen, 'Sundhets-Colleg. Aarsheretning for 1876,' p. 572.

[3] [There was a fifth in 1882.]

[4] Report in 'Sundhets-Colleg. Berättelse,' 1852, p. 30.

intervals of the epidemic outbreaks, which were all traceable to importation, those countries have been quite free from measles. In the countries of Southern Europe, such as *Roumania*,[1] *Turkey*,[2] and *Greece*,[3] the disease is prevalent to the same extent as in the other parts of the continent. Similar accounts of measles universally diffused and often coming to an outbreak, come from all the countries of *Asia*, from *Asia Minor*,[4] the *Caucasus*,[5] *Syria* and *Mesopotamia*,[6] *Persia*,[7] and *Arabia*;[8] from all the provinces of *British India*,[9] from the *East Indian Archipelago*,[10] from *Further India*,[11] *China*,[12] and *Japan*,[13] from all those regions of *Africa* of which we have any medical knowledge, such as *Egypt*,[14] *Abyssinia*,[15] *Tunis*,[16]

[1] Barasch, 'Wien. med. Wochenschr.,' 1855, No. 36; Leconte, 'Considér. sur la pathol. des prov. du Bas-Danube,' Montp., 1869, p. 45.

[2] Rigler, 'Die Türkei,' &c., ii, p. 26; Beyran, 'Gaz. méd. de Paris,' 1854, No. 22, p. 342.

[3] Olympios, 'Bayr. med. Correspondenzbl.,' 1840, No. 12.

[4] West, 'New York Med. Record,' 1869, March, p. 27.

[5] Liebau, 'Petersb. med. Jahrb.,' 1866, xi, p. 281.

[6] Tobler, 'Med. Topogr. von Jerusalem,' Berl., 1855, p. 46; Guys, 'Statist. du Paschalik d'Alep.,' Marseille, 1853, p. 63; Floyd, 'Lancet,' 1843, ii, p. 4; Robertson, 'Edin. Med. and Surg. Journ.,' 1843, July, p. 57.

[7] Polack, 'Wochenbl. der Wien. Aerzte,' 1857, p. 721.

[8] Palgrave, 'Union méd.,' 1866, No. 20, p. 308.

[9] Twining ('Clin. Illustr,' ii, p. 432) for Bengal; Cornish ('Madr. Quart. Journ. of Med. Sc.,' 1861, July, p. 84) and Shortt (ib., 1866, April, p. 221) for the Madras Presidency; Kinnis ('Edinb. Med. and Surg. Journ.,' 1851, April, p. 316) and Morehead ('Clin. Researches,' i, p. 329) for the Bombay Presidency; Don ('Bombay Med. Transact.,' 1837, iii, p. 10) for Sind; McGregor ('Diseases . . . in the N.W. Prov. of India,' p. 213) and Evans (Edinb. Med. Journ.,' 1855, Aug., p. 175) for the N. W. Provinces; Ireland (ib., 1863, Jan., p. 613) and Curran ('Dubl. Quart. Journ.,' 1871, May, p. 311) for the slopes of the Himalaya; Davy ('Account of Ceylon') for Ceylon; and various others.

[10] Waitz, 'On Diseases incidental to Children in Hot Climates,' Bonn, 1843, p. 257; Heimann, 'Krankheiten in den Tropenländern,' p. 223; Van Leent, in 'Arch. de méd. nav.,' 1867, Oct., p. 250, 1868, Sept., p. 164, 1872, Jan., p. 22; Taulier, ib., 1877, Dec., p. 401.

[11] Breton ('Considér. sur la guérison des plaies chirurg. chez les Annamites,' Paris, 1876, p. 10) for Cochin China, but said by this author to be rare there.

[12] Pearson, in 'Calcutta Med. Transact.,' 1833, vi, p. 362; Morache, in 'Annal. d'hyg.,' 1870, Jan., p. 25; Dudgeon, in 'Glasg. Med. Journ.,' 1877, July, p. 328.

[13] Gaigneron, in 'Arch. de méd. nov.,' 1866, April, p. 279.

[14] Pruner, 'Krankheiten des Orients,' p. 122; Hartmann, 'Skizzen des Nilländer.

[15] Blanc, in 'Gaz. hebd. de méd.,' 1874, No. 22, p. 349.

[16] Ferrini, 'Sul clima di Tunisi,' p. 153.

Algiers,[1] *Senegambia*,[2] the *West Coast of Africa*,[3] the *Cape*,[4] *Madagascar*,[5] *Mauritius*,[6] and *Réunion*.[7] Even the most isolated parts of the world, like *St. Helena*, have not been exempt from measles,[8] although in the reports from that island, up to date, it is admitted by everyone that the disease had not occurred there since 1807.

Measles reached the *Western Hemisphere* soon after the arrival of the first European settlers, its first appearance being in the *East Coast States* of the Union; it afterwards followed the movement of colonists westwards, showing itself in the interior or *Mississippi Valley*, where the disease spread quickly throughout Kentucky and Ohio.[9] The northern and western regions of North America had escaped down to recent times; the disease appeared first in *Oregon*[10] in 1829, and in *California*[11] in 1846, and in that year it was imported from the south into *Hudson's Bay Territory*, where it committed frightful ravages among the Indians.[12] Measles does not appear to have occurred at all in Greenland down to 1864;[13] whether it has occurred subsequently, I have not learned. There is no information as to the time of its first appearance in *Mexico* in *Central America*, or in the *West Indies*; it would appear from the accounts from these regions at the end of last century and beginning of this, that measles

[1] Guyon, in 'Gaz. méd. de Paris,' 1839, No. 46, 1842, No. 34, p. 536; Barthérand, 'Med. des Arabes;' Gaucher, in 'Gaz. méd. de l'Algerie,' 1869, No. 3, feuill. p. 34.

[2] Gauthier, 'Endémies au Sénegal,' p. 18; Chassaniol, in 'Arch. de méd. nav.,' 1865, May, p. 506.

[3] Daniell, in 'Dubl. Journ. of Med. Sc.,' 1852, Aug.; Mannerot, 'Malad. endém. à Gaboun,' p. 40.

[4] Black, in 'Edin. Med. and Surg. Journ.,' 1853, April, p. 266; Scherzer, in 'Zeitschr. der Wiener Aerzte,' 1858, No. 11, p. 166; Egan, in 'Med. Times and Gaz.,' 1873, June, p. 681, 1877, Sept., p. 355.

[5] Davidson, in 'Med. Times and Gaz.,' 1868, Dec., p. 646.

[6] Report in 'Lancet,' 1875, June, p. 865.

[7] Dutroulau, 'Traité des malad. des Européens dans les pays chauds,' Paris, 1861, p. 51.

[8] Lesson, 'Voyage méd. autour du monde,' Paris, 1829, p. 149; McRitchie, in 'Calcutta Med. Transact.,' 1835, viii, App. xxix.

[9] 'Drake, 'Diseases of the Valley of North America,' ii, p. 586.

[10] Moses, 'Amer. Journ. of Med. Sc.,' 1855, Jan., p. 38.

[11] King, ib., 1853, April, p. 389.

[12] Smellie, 'Monthly Journ. of Med. Sc.,' 1846, Dec., p. 413.

[13] Lange, 'Grönlands Syzdomsforhold,' p. 37.

was on the whole a rare disease, the epidemics being separated from one another by intervals often of twenty or thirty years, and the recurrence each time due to a fresh importation.[1]

In *Brazil*, measles appeared for the first time in the sixteenth century, coincidently with smallpox.[2] The disease is now universally distributed there and often epidemic. So also in the *River Plate States*,[3] and in *Chili*[4] and *Peru*.[5]

The disease was introduced into *Australia* in recent times. As far as the data before us justify a conclusion, it came first to the *Hawaiian Islands* in 1848,[6] and in 1854 to the *Australian continent*, whence it was imported the same year into *Tasmania*[7] and *New Zealand*.[8] Since that time, it has been repeatedly epidemic on that continent, as well as in several groups of islands, such as *Tahiti*,[9] the *Marquesas*,[10] and the *Fiji Islands*.[11] Up to 1858 *New Caledonia* had escaped altogether,[12] and the latest medical reports from that colony by Charlopin, De Rochas, and others make no mention of measles.

[1] See the accounts, for Mexico, by Stricker ('Hamb. Zeitschr. für Med.,' 34, p. 529), Porter ('Amer. Journ. of Med. Sc.,' 1853, Oct., p. 321), Bouffier ('Arch. de méd. nav.,' 1865, May, p. 533) and Heinemann ('Virchow's Archiv,' 1873, vol. 58, p. 161); for Honduras by Hamilton ('Dubl. Quart. Journ.,' 1863, Aug., p. 105); for Costa Rica by Schwalbe ('Archiv für klin. Med.,' 1875, xv, p. 344; for the West Indies by Chisholm ('Essay on the Malignant Pestil. Fever, &c.,' London, 1801, i, p. 61), Hunter ('Observ. on the Diseases of the Army in Jamaica,' German trans., Leip., 1792, p. 225) and Rufz ('Gaz. méd. de Paris,' 1857, No. 34, p. 532).

[2] Sigaud, 'Maladies du Brésil,' pp. 111, 200, 373; Rendu, 'Études méd. sur le Brésil,' p. 66.

[3] Brunel, 'Observ. méd., &c.,' p. 37; Dupont, 'Observ. méd. sur la Côte orientale d'Amerique,' Montpell., 1868, p. 14. Mastermann, in 'Dobell's Reports,' Lond., 1870, p. 382, gives an account of the disastrous epidemic of measles in the National Army of Paraguay during the Brazilian War.

[4] Account in 'Arch. de méd. nav.,' 1864, Aug., p. 103; Boyd, in 'Edin. Med. Journ.,' 1876, Aug., p. 110.

[5] Tschudi, in 'Oest. med. Wochenschr.,' 1846, p. 729; Smith, in 'Edin. Med. and Surg. Journ.,' 1840, April, p. 335; account in 'Arch. de méd. nav.,' 1864, Sept., p. 188.

[6] Gulick, in 'New York Journ. of Med.,' 1855, March.

[7] Hall, in 'Transact. of the Epidemiol. Soc.,' 1865, ii, p. 70.

[8] Tuke, in 'Edin. Med. Journ.,' 1864, Feb., p. 721.

[9] Dutroulau, 'Traité,' p. 57.

[10] Account in 'Arch. de méd. nav.,' 1865, Oct., p. 284.

[11] Account in 'Lancet,' 1875, June, p. 865, July, p. 33; Squire, in 'Med. Times and Gaz., 1877, March, p. 323.

[12] Vinson, 'Topogr. méd. de la Nouvelle Calédonie.'

§ 45. Epidemic Recurrence.

Like smallpox, measles has appeared repeatedly in far-reaching epidemics, sometimes in almost pandemic diffusion over the globe, to vanish again from the arena of national sickness after two or three years' stay. Of outbreaks of that kind during the present century—for earlier times no opinion can be formed owing to the defects of the epidemiological record—the most notable were: those of 1796-1801 in France, Germany, and England, 1801 in North America, 1807-8 in Great Britain, 1823-4 in Germany, 1826-28 in the Netherlands and Germany, 1834-36 over the greater part of Northern and Central Europe, 1842-3 in Switzerland, France, the Netherlands, Germany, and Russia, 1846-7 in Northern and Western Europe and in North America, and 1860-63 in Germany.

But an epidemic of measles may develop here and there independently of such outbreaks as those, and not unfrequently at quite isolated spots. As in the case of smallpox, many observers think that they have discovered a *periodicity* in the recurrence of these local epidemics. The intervals have been variously calculated at different places: for example, at two to three years, according to the data of Ranke[1] (Munich) and Mann (Halle);[2] at three to four years, according to Spiess[3] (Frankfurt-on-the-Main), Geissler[4] (Meran), Köstlin[5] (Stuttgart), Rigden,[6] (Canterbury), and Guys (Aleppo); at four to five years, by Macher (Gratz); and at five to six years, by Thuessink (Gröningen), Bartscher[7] (Osnabrück), and Blower[8] (Bedford). In order to arrive at the import of these figures, we have in the first place to observe that at many other places no such regular recurrence in the epidemics of measles has been found. Thus the disease

[1] 'Jahrb. für Kinderhlkd.,' 1869, ii, p. 34.
[2] 'De morbillis epid. Halis obs.,' Hal., 1848.
[3] 'Jahresbericht (Med.) der Stadt Frankfurt a. M.,' 1867.
[4] 'Vierteljahrschr. für Oeffentl. Gesundheitspflege,' 1871, iii, p. 34.
[5] 'Archiv des Vereins für wissensch. Heilkd.,' 1865, ii, p. 328.
[6] 'Brit. Med. Journ.,' 1869, April, p. 348.
[7] 'Journ. für Kinderkr.,' 1866, xlvii, p. 28.
[8] 'Assoc. Med. Journ.,' 1857, Nov., p. 924.

was prevalent at Erlangen[1] in the years 1819-25-31-39-47-52-56 ; at Christiania[2] in 1824-28-33-39-47-56-61-67 ; at Basel[3] in 1824-28-31-32-34-35-36-38-44-49-54-57-60-61-62-64-67-69-70-73 ; while in Prague[4] the intervals, in the period from 1823 to 1848, amounted to about four years, from the latter date to 1860, on the average, to two years, while since 1860 the disease has been more or less widely epidemic every year. Goldschmidt[5] states that in Oldenburg no such epidemic periodicity has ever been observed ; in Cape Town, according to Scherzer, the disease was three times epidemic in the first sixty years of the century, viz. in 1807, 1839, and 1852 ; in Martinique, according to Rufz, it was epidemic at intervals of ten years, in 1831,-41 and -51 ; in the United States of America, according to Chapman, there has been no evidence of regularity in the recurrence of measles, and the statistical information before me from Boston, New York, Philadelphia, and Baltimore plainly confirms that statement. The recurrence of the epidemic of measles at one particular place is connected neither with an unknown something (the mystical number of the Pythagoreans), nor with "general constitutional vicissitudes," as Köstlin thinks ; but it depends solely on two factors, the time of importation of the morbid poison, and the number of persons susceptible of it. The same law, accordingly, applies here as in smallpox epidemics.

§ 46. Influence of Season.

A glance at the area of distribution of measles, extending as it does over almost the whole habitable globe, shows that the occurrence of the disease is quite independent of *climatic influences*. The disease reaches as far as the morbid poison has reached ; and if some districts or tracts of country have been hitherto exempted from measles altogether or been less frequently the seat of epidemics, the reason of that is to be

[1] Küttlinger, in 'Bayr. ärztl. Intelligenzbl.,' 1860, p. 30.

[2] Reports in Eyr, and Lund in 'Fordhl. i det Norske med. Selskab i 1868,' Christ., 1869, p. 10.

[3] Hagenbach, in 'Jahrb. für Kinderhlkd.,' 1875, ix, p. 56.

[4] Popper in 'Allgem. Zeitschr. für Epidemiol.,' 1876, ii, p. 275.

[5] 'Häser's Archiv für Med.,' 1845, vii, p. 303.

sought, not in the geographical position or in the conditions of climate dependent thereon, but solely in the fact that in the one case no importation of the disease has taken place hitherto, and in the other case that the importations have been infrequent owing to the commercial intercourse being small in amount and mainly by sea.

However independent of climate, then, measles may be, whether in its geographical distribution or, as we shall see later, in the form and character of the disease; still, wherever it has occurred, the influence of *certain kinds of weather*, depending on the *seasons*, has been observed to have a marked effect upon the frequency of its outbreak and the extent of its prevalence. This influence is one and the same in all latitudes; it is everywhere the cold season in which the epidemics of measles most commonly begin, and in which they are apt to spread farthest.

There is an almost complete unanimity on this point among observers at the most diverse points of the globe. In India, according to McGregor, Morehead, and others, the season for measles falls in the months from February to April, or in the cold season; the same is true for Brazil, according to Sigaud and Rendu; in Persia and Egypt, as Polack and Pruner inform us, the disease is mostly prevalent in spring and autumn; Rigler makes the same statement for Turkey; at the Cape almost all the epidemics have been observed in the autumn from April to June (Scherzer); in North America, the sickness begins mostly towards the end of winter and lasts through the spring (Drake); and that is also the statement made for Switzerland by Hoffmeister,[1] for Prague by Popper, and for Roumania by Leconte. Of 30,836 fatal cases of measles in England and Wales in the years 1838-1840 and 1849-1853, there occurred 8106 in the months from January to March, 8907 from April to June, 6610 from July to September, and 7213 from October to December. The following is a table of 530 epidemics of measles in temperate latitudes (Europe and North America), the duration of which is accurately given:

[1] 'Schweiz. Zeitschr. für Med.,' 1849, p. 471.

In Autumn	32	Total in the colder months —339.
In Autumn and Winter	52	
From Winter to Spring	15	
In Winter	62	
In Winter and Spring	74	
From Winter to Summer	27	
In Spring	77	
In Spring and Summer	81	Total in the warmer months —191.
From Spring to Autumn	26	
In Summer	38	
In Summer and Autumn	36	
From Summer to Winter	10	

In 213 of the same epidemics, of which there is mention of their coming to a height, that point was reached forty-eight times in autumn, fifty-nine times in winter, seventy-six times in spring, and thirty times in summer.

While there can be no doubt, then, that the season of the year, or, in fact, the kind of weather associated with the colder months, exerts a certain influence on the rise and spread of measles epidemics, the question of how this influence is exerted still remains unsolved. That this prevalence of the disease in the colder seasons is not in consequence of a change in the habits of living associated therewith,—the crowding together in close rooms, and the facilities thereby afforded for communicating the disease,—may be inferred from the fact that the same degree of dependence on the season of the year obtains as much in the tropics as in high latitudes,—in India, in Southern China, and in Brazil, countries where crowding in close rooms can hardly be taken into serious account as a factor in the etiology.

§ 47. Mildness or Severity of Type.

Just as little does the *conformation or character* of the epidemics of measles appear to depend upon climatic or seasonal conditions, or on the weather. There are, generally speaking, no real differences to be made out in the course which measles runs at the various points of its large area of distribution; everywhere the mucous membrane of the respiratory organs is, next to the skin, the chief focus of the disease as localised, and even the (rarer) implication of the intestinal mucous membrane does not appear to be, *cæteris*

paribus, more frequent or more severe in the tropical, or subtropical, zones than in higher latitudes.

The character of the disease is in the great majority of cases as mild, and its course as favorable, in tropical or subtropical as in temperate climates. On this point there is much agreement in the accounts of Morehead, McGregor, Twining, Huillet,[1] and others for India, of Heymann and Waitz for the East Indies, of Dudgeon for China, Polak for Persia, Gauthier for Senegambia, Schwarz[2] for Rio Janeiro, Boyd for Chili, and of others for still other countries. Accordingly, when we hear from some observers, such as Curran for the southern slopes of the Himalaya, Hamilton for Honduras, and Davidson for Madagascar, of a peculiarly malignant form of the disease, we must look for an explanation of that fact, not by any means in the circumstances of the climate, but chiefly in certain habits of living to be afterwards mentioned, which make themselves felt most wherever the principles of hygiene and of rational therapeutics have been most grievously sinned against.

Statistics give no support to the notion that the season of the year or the kind of weather has in itself any real effect on the character of the epidemic. Among the accounts before me of epidemics of measles in temperate latitudes, I find 285, in which somewhat definite statements are made regarding the mildness or severity of type. They divide themselves as follows:

	In Autumn.	In Winter.	In Spring.	In Summer.
Of a mild type	55	58	74	53
Of a severe type	10	12	13	10
Proportion of severe to mild	1 to 5·5	1 to 4·9	1 to 5·7	1 to 5·3

The proportion of mild to malignant epidemics is, therefore, tolerably uniform in all seasons; and this result is fully borne out by the numerous individual observations made for those epidemics which extend over longer periods. The severity of epidemics of measles depends in great part on the intensity of the affection in the organs of respiration and digestion; and thus, from the fact that the epidemics in summer and winter are more often unfavorable in character-

[1] 'Arch. de méd. nav.,' 1868, Jan., p. 24.

[2] 'Zeitschr. der Wien. Aerzte,' 1858, p. 579.

than those of spring and autumn, the conclusion might be drawn *à priori*, that the severity of the summer epidemics depends for the most part upon the more considerable implication of the intestine (catarrh and dysentery), and of the winter epidemics upon the intensity of the process in the respiratory organs (croup, capillary bronchitis, broncho-pneumonia). But this assumption is not warranted by the facts.

Thus Pommer[1] says, of the 1827 epidemic at Heilbronn, that, while in the summer time it had a severe character from being complicated with croup, its course in the autumn, and on to its termination in December, was altogether more favorable. In the epidemic from January to autumn, 1837, at Ahrweiler (Gov. Depart., Coblenz) the type of the disease was very favorable in the spring months, and it was not until the setting in of the dry and hot weather of summer that it became dangerous from croup complications.[2] The epidemic of measles at Brussels in the spring and summer of 1837 owed its character for malignancy to the severe pneumonic attacks in the summer months,[3] and the circumstances were repeated the same year at Paris,[4] without the influence of the weather in either case being chargeable as the cause of the pneumonia. In the epidemic that prevailed at Dublin in 1844, in mild summer weather, the somewhat high rate of mortality among the sick was mostly due to croup.[5] On the other hand, there have been many epidemics of measles observed within the temperate zone, which took on a malignant character in consequence of a severe intestinal affection; but in none of them did the season of the year or the kind of weather serve to explain this modification of the morbid process; as examples, may be mentioned the epidemic in the autumn of 1821 at Salem (Mass.),[6] a similar epidemic in 1832 at Berlin,[7] and in 1837 at Hamburg,[8] and, in winter, 1837-38, at many parts of Würtemberg,[9] 1846-47 at Hamburg,[10] 1848 at Jackson (Miss.),[11] and 1853 in the department of the Haut-Saône.

There is, however, no doubt that states of the weather, in so far as they affect definite groups of organs, cause these to

[1] 'Salzb. med.-chir. Ztg.,' 1828, No. 28, ii, p. 30.
[2] 'Sanitätsber des Rhein. Med.-Colleg. für das Jahr 1837,' p. 29.
[3] Daumerle in 'Bullet. méd. Belge,' 1839, ii, p. 33.
[4] Account in 'Gaz. méd. de Paris,' 1837, No. 25.
[5] Lees in 'Dubl. Quart. Journ.,' 1844, Sept.
[6] Pierson in 'New Engl. Journ. of Med.,' 1822, xi, p. 122.
[7] Lieber in 'Casper's Wochenschr. f. Heilkde.,' 1833, i, p. 264.
[8] Warburg in 'Hamb. Zeitschr. für Med.,' ix, p. 10.
[9] Account in 'Württemb. med. Correspondenzblatt,' 1841, xi, 187—189.
[10] Stahlmann in 'Hamb. Zeitschr. für Med.,' xxxvi, p. 18.
[11] Ferrar in 'Southern Med. Reports,' 1850, i, p. 354.

become the *locus minoris resistentiæ*, and therefore the seat of the diseased action; and, as a matter of fact, the process of measles in tropical regions appears from the statements of Morehead (India), Ferrini (Tunis), Daniell (West Coast of Africa), and Sigaud (Brazil), to be more often accompanied by severe affection of the intestine than in temperate latitudes. But in those regions, also, this configuration of the disease obtains at times when weather influences cannot well be regarded as the cause of it; as, for example, in the severe epidemics of measles in Java in the winter (December—January) of 1849 and 1850,[1] and at Bombay in the months of March and April, 1857.[2]

There is not the very smallest ground for the belief that local conditions of the soil exert an influence on the rate of diffusion or on the type of measles. When Fuchs[3] says that "in the north, and in elevated districts, the inflammatory form occurs, while in the south and in flat stretches of coast it is more usually the asthenic and putrid form," he omits at the same time to give his proof for this statement; and when he adds: "England, Holland, and many parts of the French coast are noted for having been long the scene of the most malignant epidemics of the disease, and the frequent occurrence of putrid measles in marshy lands leads one to think that miasmata may have helped to generate this worst of all varieties"—I can nowhere find any facts to justify the assertion. Thuessink, to whom we owe an excellent work upon the occurrence of measles in the Netherlands in recent times, does not say one word about any malignant character of the disease, such as has been attributed to that country; and the epidemic of measles in 1847-49, which was diffused over the whole country, ran a course that was almost uniformly favorable. The little influence that this factor has on the character of the disease is further shown in the fact that it is precisely in the great marshy districts of many tropical regions that measles has, generally speaking, a very mild course.

[1] Broekmeyer, in 'Arch. de méd. nav.,' 1868, Dec., p. 415.

[2] Carter, in 'Bombay Med. Transact.,' 1859, n.s., iv, p. 253.

[3] 'Die Krankhaften Veränderungen der Haut,' Abt. iii, p. 361.

§ 48. Severity of Type due to Neglect of the Sick.

In considering the reason why some epidemics of measles should have had a malignant type, great stress, in my opinion, should be laid on *mistakes in dieting and in therapeutic treatment*. Without doubt it is here that we have the explanation of the fact that the disease in past centuries had a much more unfavorable type than in recent times. In forming an opinion, however, on this point, we should bear in mind that many epidemics of measles adduced in evidence from the eighteenth century were, in fact, outbreaks of scarlet fever. But there still remain a considerable number of true measles-epidemics of that period, whose malignant character was due in the last resort, as the chroniclers themselves admit, to the way in which the sick were treated. Even for many of the epidemics of the last thirty or forty years, remarkable for their very considerable mortality, it could be shown that reactionary dietetic and therapeutic practices gave the epidemic its malignant character. The importance of that factor in the causation comes out in the clearest way in those epidemics of measles which, springing up among uncivilised peoples, have run a disastrous course in the absence of all rational treatment of the sick.

Classical examples of this are furnished by the epidemic of 1749 among the natives on the banks of the Amazon, where the number of those that died of the sickness was reckoned at 30,000, whole tribes having been cut off;[1] also in Astoria in 1829, where nearly one half of the natives fell victims to the disease;[2] among the Indians of Hudson's Bay Territory in 1846;[3] among the Hottentots at the Cape in 1852;[4] among the natives of Tasmania[5] in 1854 and 1861; and in Mauritius and the Fiji Islands in 1874. Concerning the two last mentioned epidemics, both of them disastrous, it is stated in the report:[6]—"The great mortality has been in large measure due to the fact that the sick were exposed to the most unfavorable conditions. Unprotected from exposure, unattended and untreated, chiefly in consequence of their own unhappy prejudices, every complication of the disease must have been invited and rendered intense; in accordance with this view, we find that those classes of the native population over whom adequate supervision could be exercised have suffered slightly." Smellie mentions facts of the same kind in the destructive epidemic of 1846 among

[1] Sigaud, p. 111. [2] Moses. [3] Smellie. [4] Scherzer. [5] Hall.
[6] 'Lancet,' 1875, June, p. 865.

the natives of Hudson's Bay Territory; of all those who were received into Fort York, and who there received medical treatment, not one died. In the account given by Squire[1] of the frighful epidemic of measles in the Fiji Islands, which was known to have been introduced from Sydney by the retinue of King Kakobau, and which carried off 20,000 of the natives, or one fourth to one fifth of the whole population of the Fiji group, we find the following:

"The favorable progress of the early native cases negatives the idea of any special proclivity. Dr. Cruikshank, who treated 143 of the native constables, reports nine deaths, most of these resulting from evasion of needful precautions. Later in the epidemic, when it is said to be like plague, and that the people, seized with fear, had abandoned their sick, only one death occurred among a number of cases treated in separate rooms with fair attention. . . . The people chose swampy sites for their dwellings, and whether they kept close shut up in huts without ventilation, or rushed into the streams and remained in the water during the height of the illness, the consequences were equally fatal. The excessive mortality resulted from terror at the mysterious seizure, and [from] the want of the commonest aids during illness. . . Thousands were carried off by want of nourishment and care, as well as by dysentery and congestion of the lungs. . . We need invoke no special susceptibility of race or peculiarity of constitution to explain the great mortality."

But it is not necessary that we should seek in so distant regions and among uncivilised peoples for proofs of the disastrous influence of unfavorable hygienic conditions upon the type of epidemics of measles on a large scale. In the epidemic which prevailed in 1866 among the Confederate troops during the American Civil War, there were 1900 deaths out of 38,000 cases of sickness. In the official report,[2] it is stated that "the disease resembled ordinary measles in adults, except when aggravated by the effects of crowd, poisoning, or other depressing influences;" in two large hospitals, the mortality amounted to 20 per cent. of the sick. In Paris during the siege (January, 1871), out of 215 of the Garde Mobile who took measles, 86, or 40 per cent., died; and the mortality reached very nearly the same figure among the French troops who returned to Paris after the Italian war, 40 out of 125 cases dying in one hospital, (whose sanitary condition was bad) with severe intestinal symptoms.[3] Speaking of the disastrous epidemic of measles

[1] 'Med. Times and Gaz.,' 1877, March, p. 323.

[2] 'Med. History of the Rebellion,' Philad., 1865, p. 127.

[3] Laveran, in 'Gaz. hebdom. de méd.,' 1861, No. 2.

in the National Army of Paraguay, Masterman[1] says: "At the beginning of the Brazilio-Paraguayan war, an epidemic of measles swept off nearly a fifth of the National Army in three months, not from the severity of the disease, for I treated about fifty cases in private practice without losing one, but from want of shelter and proper food."

I will not say that these considerations enable us to understand completely why some epidemics of measles are of a severe type; there may be still other factors, acting on the physiological disposition of the people in a given locality, or there may be even a concentration of the morbid poison, determining the unfavorable type of the epidemic. But the favourite phrase "constitutio epidemica" does not help us at all in the elucidation of the question.

§ 49. The Specific Poison of Measles.

It will hardly be questioned nowadays that a *specific poison* underlies the disease of measles, that this poison reproduces itself within the diseased organism, and that the spread of the disease from person to person and from place to place takes place solely by the conveyance of the poison. As to the *nature of the poison*, which, as it is capable of reproduction, must at all events be taken to be an organic body or to issue from a special organic body, we have investigations by Salisbury,[2] Klebs,[3] and others; but the result of those inquiries has not as yet been confirmed. We are also without any certain information, up to the present, as to organ or organs of the sick person by which the poison is eliminated, whether by the skin only or by the mucous membranes as well. So much may be safely concluded: that the virus passes from the sick into the atmosphere around, which thereupon becomes a carrier of the infective substance within a certain short range, not ascertainable more accurately; or that it is deposited from the air upon articles (linen, clothes, &c.) which have been used by the patient or have been in

[1] L. c., p. 384.

[2] 'Amer. Journ. of Med. Sc.,' 1862, July 17, Oct., p. 387.

[3] 'Verhandl. der Würzb. Phys.-med. Gesellsch.,' 1874, n. s. vi, Sitzungsbericht, vii.

his immediate proximity; and that it may be brought, clinging to these, into other rooms, there to give rise to new foci of infection.

All that we may infer from observed facts as to the tenacity of life of the virus of measles, has been already stated in the beginning of this chapter. The *area of the disease* extends, as we have seen, to wherever the morbid poison has been imported, and the disease lasts as long as the poison can find a soil in which to multiply—in the bodies of those who are susceptible. This susceptibility to the virus of measles is proved by the geographical distribution of the disease, to be uniformly shared by the whole of mankind, of whatever *races* or *nationalities*. And if, among the coloured peoples, measles puts on its severest forms and leads to disastrous results exceptionally often, the reason of that does not lie in their physiological peculiarities, but mainly, as we have seen, in the unfavorable hygienic conditions amidst which they live.

CHAPTER VI.

SCARLET FEVER.

§ 50. Historical Notices Defective.

The earliest information about scarlet fever, to which any historical certainty appertains, goes hand in hand, as we have seen, with the information about measles. Both morbid processes were discussed in common, under various designations, by the mediæval physicians as well as by those of the earlier centuries of the modern period; and as late as the 17th century, after the special features of the scarlatinal process had come to be recognised, many physicians clung to the opinion that it was only a modification of measles. It was not until the middle of the eighteenth century that a perfectly clear understanding on this point was arrived at; but there was introduced into the doctrine of scarlet fever at the same time a new error, which makes itself heard even at the present day. One-sided emphasis placed on the inflammatory process in the throat, which so often occurs in scarlatina, led to its being confused with angina maligna (diphtheritis of the throat), and the papular or vesicular efflorescences that occur not unfrequently in cases where the cutaneous exanthem is severe, led to its being confounded with miliary fever. Those errors are reflected in the historical inquiries of Most,[1] Fuchs,[2] Hecker,[3] and others; I shall postpone the discussion and correction of them until I come to treat of the history of malignant sorethroat.

The origin and native habitat of scarlatina are questions that do not admit of an answer; we are equally unable to

[1] 'Versuch einer kritischen Bearbeitung der Geschichte des Scharlachfiebers, &c.,' 2 vols., Leipzig, 1826.

[2] 'Historische Untersuchungen über Angina maligna,' Würzb., 1828.

[3] 'Geschichte der neueren Heilkunde,' Berlin, 1839, 200—274.

decide as to the time when the disease first attained its general prevalence on the *Continent of Europe*. It was at any rate long before the period from which we derive the earliest medical accounts of scarlet fever; and the statements that it first appeared in England and Scotland in 1661,[1] at Berlin in 1716,[2] at Florence in 1717,[3] and in Denmark in 1740,[4] are most probably to be accounted for by the circumstance that the disease, now that it had been recognised by its special features, had then attracted the particular attention of the profession and the public. When and where scarlet fever first appeared on *Asiatic* and *African soil* cannot be made out even approximately. On the continent of *North America* it was probably in 1735, and in New England, that scarlatina first obtained a footing; the first occurrence of the disease in *South America* falls in the years from 1829 to 1831. On the *Australian continent* and in *Polynesia* it was first seen in 1847-48; of which more in the sequel.

The oldest notice relating probably to an epidemic of scarlatina dates from Sicily, 1543;[5] next come the well-known accounts by Döring[6] for Breslau, and by Sennert[7] for Wittenberg in the year 1627; then the report by Winsler[8] for Brieg, 1642, and that by Fehr[9] for Schweinfurt, 1652; further, the works of Sydenham,[10] who contributed materially to the correct understanding of the disease, and of his

[1] Sibbald, 'Scotia Illustrata,' Edin., 1684, p. 55.

[2] Gohl, 'Act. med. Berolin,' Dec. i, vol. 1, p. 30, ii, p. 4.

[3] Calvi, in Roncalli Parolino's 'Europae Medicina,' Brix., 1747, p. 333. This author, who refers to Sydenham, and gives a good description of the disease, remarks: "Febris prima epidemia triginta circiter abhinc annis Florentiae fuit."

[4] Wernicke, 'Spec inaug. de febri scarlatina,' Hafn., 1760, p. 23; "constat, illum [morbum] his in terris vix cognitum fuisse, sed primo intra 1740 ad 1750 hic inclaruisse."

[5] Compare Corradi ('Annali delle epidem. occorse in Italia,' ii, p. 287), for a reference to the treatise by Paulus Restifa ('Epistol. med. ad Franciscum Bissum,' &c., Messina, 1589), who gives an account of this epidemic.

[6] In Sennert's 'Epistol.,' Cent. i, ep. 88, Opp. Lugd., 1676, vi, p. 620.

[7] Ib., Cent. ii, ep. 20, l. c., p. 644.

[8] 'Ephemer. nat. cur.,' Dec. i, Ann. 6 et 7, 1675-76, Obs. 42.

[9] 'Auchora sacra, &c.,' Jenæ, 1666, p. 90.

[10] 'Observ. med.,' Sect. iv, cap. ii, Genev., 1736, i, p. 162. It is probable that Sydenham was not the first to use the term "febris scarlatina;" in the 'Monum. stor. Moden.' of Lancellotti, i, 208, 382, as Corradi points out, there is a reference under the year 1527: "Che nella primavera, mentre puti (putti) assai hano li

countryman Morton ;[1] and subsequent accounts from France[2] (1712), from Sweden[3] (1741), and from the Netherlands.[4] Among the best works on scarlet fever of that period, are those of Storch,[5] based upon observations—he gives an account of more than 190 cases—made at Gotha in the years from 1717 to 1740.

§ 51. Present Geographical Distribution.

By far the largest *area of distribution* of scarlet fever is met with on *European soil.* In *Germany, France,* the *Netherlands, England,*[6] and the *Scandinavian* kingdoms, this disease is one of the chief factors in the statistics of sickness and mortality. In *Russia* also, it appears to be somewhat widely prevalent.[7]

That the most northern as well as the most southern countries of this part of the globe enjoy no real immunity from the sickness is proved, on the one hand by the account of Schleisner[8] for *Iceland,* and, on the other hand, by those of Menis[9] and De Renzi for *Upper* and *Lower Italy ;* of

varoli, altri muojono da male da scarlatina." It is, however, an open question whether "febris scarlatina" is really meant here.

[1] 'Pyretologia,' Cap. v, Genev., 1696, p. 28.

[2] In 'Journ. de méd.,' 1763, June, p. 551.

[3] Rosenstein, 'Anweisung zur Kenntn. und Kur der Kinderkr.,' Gött., 1768, 417.

[4] De Haen, 'Thes. sist. febr. divis.,' Vindob., 1760, p. 25 ; 'Ratio med.,' i, p. 96.

[5] 'Pract. und theoret. Tractat. v. Scharlachfieber, &c.,' Gotha, 1742.

[6] In England and Wales the annual mortality from scarlet fever, on the average of eight years from 1848 to 1855, was one twenty-fifth of the total mortality, and there were many years in which it was as much as one twentieth (Farr, in 'Annual Report of the Registrar General,' 1857, p. 180).

[7] There is a noteworthy remark by Ucke ('Das Klima und die Krankheiten der Stadt Samara,' Berl., 1863, p. 198), that scarlet fever is so extremely rare in Samara that the public has no knowledge whatever of the disease, and that there is therefore no colloquial name for it.

[8] 'Island undersögt, &c.,' p. 53. The disease was epidemic there in 1797 and 1827, probably also as early as 1669 and 1776. In 1848 a few cases were observed ('Sundhedskoll. Forhandl. for aaret., 1849,' p. 9) ; while Finsen ('Sygdomsforholdene i Island,' p. 47) did not see a single case during a ten years' residence on the island (1856-66). The Faröe Islands, according to Panum, had quite escaped the disease up to 1847 ; I am not acquainted with any later data as to scarlet fever there.

[9] 'Saggio di topogr. stat.-med. della provincia di Brescia, &c.,' Bresc., 1837, i, p. 154.

Oppenheim[1] and Rigler[2] for *Turkey;* of Olympios[3] for *Greece,* of Moris[4] for the island of *Sardinia,* and of Zulati[5] and Jenner[6] for the *Ionian Islands* and *Malta.*

A highly striking contrast to this frequency of scarlet fever in Europe, especially in its Central and Northern parts, is presented by its very scanty diffusion hitherto in *Africa* and *Asia.* It is rare all over the East, and especially so in *Egypt* (says Pruner[7]), being seen mostly in sporadic cases of a mild type, while in the south of Egypt the disease does not appear to occur at all.[8] There are accounts to the same effect, of the amount and character of the disease in *Abyssinia,*[9] *Tunis,*[10] *Senegambia,*[11] the *Cape,*[12] and *Madagascar.*[13] It is only in *Algiers* that scarlet fever is said to be somewhat common,[14] and to occur, in fact, not unfrequently in malignant epidemics.[15] In the *Azores* it has often been epidemic.[16] Into *Madeira* it was introduced for the first time at the beginning of this century;[17] there was no return of it between the years 1814 and 1824; and in the medical reports from the island for the subsequent period,[18] it is spoken of as a disease occurring seldom, and then of a mild type.

[1] 'Ueber den Zustand der Heilkunde in der Türkei,' Hamburg, 1833, p. 56.

[2] Op. cit., ii, p. 23.

[3] 'Bayr. med. Correspondenzblatt,' 1840, p. 178.

[4] In De Marmora's 'Voyage en Sardaigne, &c.,' Paris, 1826.

[5] 'Giorn. di med.,' Venezia, 1764, ii, p. 224.

[6] 'Sketches of the Med. Topogr. of the Mediterranean, &c.,' Lond., 1830.

[7] 'Krankh. des Orients,' p. 120.

[8] Hartmann ('Skizze der Nil-länder,' p. 419) mentions, on hearsay evidence, the occasional occurrence of epidemics in the southern countries of the Nile basin.

[9] Blanc in 'Gaz. hebd. de méd.,' 1874, No. 22, p. 349.

[10] Ferrini, p. 154.

[11] Chassaniol in 'Arch. de méd. nav.,' 1865, May, p. 506; Gauthier, 'Des Endémies au Sénégal,' Paris, 1865, p. 18.

[12] Scherzer in 'Zeitschr. der Wiener Aerzte,' 1858, p. 156; Egan, in 'Med. Times and Gaz.,' 1873, June, p. 682.

[13] Borchgrevink in 'Norsk Magaz. for Laegevidensk,' 1872, p. 247.

[14] Guyon in 'Gaz. méd. de Paris,' 1839, No. 46; Gaucher in 'Gaz. méd. de l'Algerie,' 1869, No. 3, p. 34.

[15] Claudot in 'Rec. de mem. de méd. milit.,' 1877, p. 193.

[16] Nogueira in 'Jorn. da societ. das scienc. med. de Lisboa,' xxiii.

[17] In 1806, according to Gourlay, 'Med. and Phys. Journ.,' 1811, May, p. 430. See also Heineken in 'Lond. Med. Reposit.,' 1824, July, p. 14.

[18] Kämpfer in 'Hamb. Zeitschr. für die ges. Med.,' 1847, xxxiv, p. 166.

On *Asiatic* soil, the coast of *Asia Minor* appears to be the only region which is frequently visited by scarlatina in its severe forms.[1] In *Syria*,[2] *Mesopotamia*,[3] *Persia*,[4] and *Arabia*,[5] the disease is seen only in rare sporadic cases, if it occur at all; and that is also the state of matters in the *East Indies*,[6] in *Further India*,[7] and most probably also in *British India*. As regards the last, some observers, such as Chevers[8] for Bengal, Rhude[9] for Tranquebar, Huillet[10] for Pondicherry, the authors of reports from Madras,[11] Collins[12] for the plateau of the Deccan, Morehead[13] for Bombay, and Evans[14] for Mirzapore, declare that neither has any case of scarlet fever come under their own notice nor has any such case been proved to have occurred in India at all; while others, particularly Hogg[15] and a few practitioners in Lower Bengal,[16] remark that the disease has often been imported into India, but has never become epidemic there, having been always limited to a few mild cases among European or Eurasian children. There are unquestionably mistakes in diagnosis underlying these statements as to the isolated occurrence of scarlatina in India, especially the mistaking of dengue for it; and, considering this, Milroy[17] comes to the conclusion that the disease is clearly proved to have

[1] Pruner, l. c.

[2] Pruner; Tobler ('Zur med. Topogr. v. Jerusalem,' Berl., 1855, p. 46). Robertson ('Edin. Med. and Surg. Journ.,' 1843, July, p. 57) makes no mention of scarlet fever beside smallpox and measles.

[3] Ffloyd, 'Lancet,' 1841.

[4] Polak, in 'Wiener med. Wochensch.,' 1855, No. 17.

[5] Palgrave, in 'Union méd.,' 1866, No. 20, p. 308.

[6] Heymann ('Krankh. in den Tropenländern.,' Würzb., 1855, p. 224), during a long residence in Java, saw only a few sporadic cases.

[7] Dawson ('Philad. Med. Examiner,' 1852, May) says that he had heard of scarlet fever occurring in Burmah among the children of the English missionaries; Breton (l. c.) mentions that the disease is very rare in Anam.

[8] 'Med. Times and Gaz.,' 1879, Jan., p. 4.

[9] 'Bibl. for Laeger,' 1831, April, p. 263.

[10] 'Arch. de méd. nav.,' 1868, Jan., p. 25.

[11] In 'Report of the Sanitary Commissioners for Madras,' 1869.

[12] 'Ind. Annals of Med. Sc.,' 1860, Nov., p. 5.

[13] 'Researches on Diseases in India,' i, p. 360.

[14] 'Edin. Med. Journ.,' 1855, Aug.

[15] 'Med. Times and Gaz.,' 1876, Sept., p. 253.

[16] 'Indian Med. Gaz.,' 1871, Oct., p. 2.

[17] 'Transact. of the Epidemiol. Soc.,' 1865, ii, p. 156.

existed at one point only, viz. Colombo in Ceylon, and there merely to a slight extent and in a mild form. In the Southern and South-Eastern coast towns of *China*, it occurs very rarely, if at all;[1] I am unable to decide what ground there is for the assertion of Morache[2] that the disease has been often epidemic in Pekin. In *Japan*, it is said by Wernich[3] to be quite unknown.

Scarlatina reached *Australia* and *Polynesia*, first in the beginning of 1848; the disease broke out almost simultaneously in *Tahiti*,[4] *New Zealand*,[5] and *Tasmania*,[6] but everywhere to a limited extent and in a very mild form. In 1853 it appeared afresh in Tasmania, and in 1854 in New Zealand,[7] reaching the Australian continent simultaneously. More special details of the scarlet fever in that part of the world have not come under my notice; there is a solitary reference to a malignant epidemic in Melbourne in the year 1876.[8] With the exception of Tahiti, the islands of Polynesia appear to have escaped the disease hitherto.

The first appearance of scarlet fever on the soil of *North America* dates from 1735; according to Douglas[9] and Colden,[10] the disease broke out first in Kingston, Mass., it followed quickly in Boston and other places near, and a little later in New Hampshire, overran the whole of the New England States in the course of the next few years, came next to New York and to Philadelphia in 1746, as Morris[11] asserts on the authority of a manuscript note by Kearsley, and travelled thence,

[1] 'Armand, in 'Gaz. méd de Paris,' 1861, No. 17, p. 201; Rochefort, in 'Arch. de méd. nav.,' April, p. 241; Dudgeon, in 'Glasgow Med. Journ.,' 1877, July, p. 328.

[2] 'Annal. d'hyg.,' 1870, Jan., p. 55.

[3] 'Deutsche med. Wochenschr.,' 1871, No. 9, p. 101.

[4] Account in 'Arch. de méd. nav.,' 1865, Oct., p. 283.

[5] Thomson, in 'Brit. and For. Med.-Chir. Rev.,' 1855, April.

[6] Hall, in 'Transact. of the Epidemiol. Soc.,' 1865, ii, p. 72.

[7] Tuke, in 'Edin. Med. Journ.,' 1864, Feb., p. 721.

[8] 'Brit. Med. Journ.,' 1876, May, p. 609.

[9] 'The Practical History of a New Epidemical Eruptive Miliary Fever,' Boston, 1736. Reprinted in the 'New England Journ. of Med.,' 1825, Jan., p. 1. Fuchs, Hecker, Häser, and others who mention this paper in writing of angina maligna (diphtheria), can hardly have read it in the original, otherwise they would not have taken the disease to be anything but typical scarlet fever.

[10] 'Lond. Med. Observ. and Inquiries,' 1754, i, p. 211.

[11] 'Lectures on Scarlet Fever,' Philad., 1851.

as it appears, along the Atlantic coast to South Carolina, where it is referred to by Chalmers,[1] towards the end of the eighteenth century, as being certainly of rare occurrence. In 1784 scarlet fever reappeared in the Northern States, and in 1791–93 it penetrated for the first time into the interior of the continent, particularly into Kentucky and Ohio.[2] During the present century it has become general, from *Canada* (where it was especially prevalent in 1843, from Toronto onwards, over a wide area)[3] to the Gulf Coast States. In the Southern States, whence we have epidemiological data for the year 1821 from Arkansas[4], 1832 from Augusta, Ga.,[5] 1833 and 1843 from Alabama,[6] 1847 from New Orleans,[7] and 1854 from Raleigh, N. Car.,[8] the occurrence of scarlet fever, according to the unanimous opinion of observers, is much rarer than in the Northern. It appears from Sozinsky's interesting statistical notice[9] of the distribution and amount of the disease, based on the results of the U. S. census of June, 1870, that, while the yearly mortality was from 30 to 160 per 100,000 inhabitants in the New England States, New York, New Jersey, Pennsylvania, Maryland, Ohio, Virginia, Indiana, Illinois, Michigan, Wisconsin, and Iowa, it fell to between 1·0 and 9·4 in Tennessee, North and South Carolina, Georgia, Alabama, Mississippi, Louisiana, Florida, Arkansas, and Texas. *Greenland* has escaped scarlet fever hitherto,[10] with the exception of a single case in 1848, a child of the district physician Rudolph. It has shown itself now and then in *Newfoundland*;[11] but there is no information about it from Nova Scotia, New Brunswick, and Hudson's Bay Ter-

[1] 'Account of the Weather and Diseases of S. Carolina,' Lond., 1776, vol. ii (German. transl., 1796, ii, p. 209).

[2] Drake, l. c., ii, p. 599.

[3] Stratton, in 'Edin. Med. and Surg. Journ.,' 1849, April, p. 269.

[4] Huntt, in 'Amer. Med. Recorder,' 1822, v, p. 277.

[5] Robertson, in 'Amer. Journ. of Med. Sc.,' 1834, Feb.

[6] Basset, in 'South. Med. Reports,' 1850, i, p. 266; Bates, ib., p. 313.

[7] Rhodes, ib., p. 239.

[8] McKee, in 'Transact. of the Carol. State Med. Assoc.,' 1856.

[9] 'Philad. Med. and Surg. Reporter,' 1880, Jan., p. 68.

[10] Account in 'Sundhedscoll. Forhandl. for aaret 1848,' p. 7; Lange, 'Bemaerkninger om Grönlands Sygdomsforhold.' Kjöbenh., 1864, p. 37.

[11] Gras, 'Quelques mots sur Miquelon,' Montp., 1867, p. 30; Anderson, in 'Dobell's Reports,' 1870, i, p. 365.

ritory. On the Pacific coast of North America it was seen first in 1851 in *California*;[1] at the outset it occurred only in isolated cases and in a very mild form, but subsequently there have been epidemics of malignant scarlatina. Its epidemic occurrence in *Mexico* is mentioned by Stricker[2] and by Robredo;[3] on the other hand, Heinemann states that not a single case of scarlet fever came under his notice during a six years' residence at Vera Cruz.[4] There are only scattered notices of the disease from *Central America*; Hamilton[5] states that it is rare in *Honduras*, but of a malignant type; Schwalbe[6] mentions a severe epidemic in *Costa Rica* in 1856.

I have had no means of finding out when scarlet fever first reached the *West Indies*. It is remarkable that the medical accounts from these colonies in the last century are quite silent about the disease; the earliest mention of it is by Savarésy,[7] and concerns a very mild epidemic in March, 1802, in Martinique; Rufz saw the disease there also in 1835,[8] but it had not recurred from that time down to 1856. Forström[9] says that it is not uncommon in Guadeloupe, but that St. Bartholomew had quite escaped it up to that time (1812); it first became epidemic in the latter, as Cock states,[10] in 1829-30. These few notices, and an account by Pop[11] of a severe epidemic in Curaçao, are all that I have been able to collect about the disease in those regions. In New Providence (*Bahamas*), an imported case from America was observed in 1845; Duncome[12] communicates this fact, with the remark that nothing was known of the disease having occurred on the islands during the previous forty years.

It was about 1830 that scarlet fever began to be generally

[1] Praslow, l. c., p. 55; Gibbons, 'Annual Address delivered before the San Francisco State Med. Soc.,' 1857, p. 10.

[2] 'Hamb. Zeitschr. f. Med.,' 1847, vol. 34, p. 529.

[3] 'Periodico de la Acad. de med. de Méjico,' 1838, Sept.

[4] 'Virchow's Archiv,' 1873, vol. 58, p. 161.

[5] 'Dublin Quart. Journ. of Med. Sc.,' 1863, Aug.

[6] 'Archiv für klin. Med.,' 1875, xv, p. 3.

[7] 'De la fièvre jaune,' Naples, 1809, p. 23.

[8] 'Arch. de méd. nav.,' 1869, Aug., p. 136.

[9] 'Svenske Läk. Sällsk. Handl.,' 1812, iv, p. 231.

[10] 'Edin. Med. and Surg. Journ.,' 1832, Jan., p. 28.

[11] 'Nederl. Tijdschr. voor Geneesk.' 1859, iii, p. 212

[12] 'Lancet,' 1846, March.

diffused over *South America*. According to Brunel,[1] it was once prevalent before in 1796 in the *River Plate States*, and it broke out anew in 1831 (as Sigaud[2] also mentions) in Buenos Ayres, whence it spread to Monte Video, and in 1832 reached the southern and central provinces of *Brazil* (Rio Grande, Santa Catarina, São Paulo, Minas) and ultimately Rio Janeiro. Since that time, the Argentine Republic and Brazil have been visited frequently by severe epidemics.[3] In *Chili* also, its first appearance[4] corresponds to the time of this general diffusion of the disease on the eastern side of South America; and it has been often observed subsequently in that country in epidemics that have been not unfrequently of a malignant type. There are accounts to the same effect, by Tschudi[5] and by Smith,[6] of its prevalence in *Peru*.

§ 52. Peculiarities of Scarlatinal Epidemics as compared with those of Smallpox and Measles.

A glance at the map of its geographical distribution, as here sketched in general outlines, shows that the *area of diffusion* of scarlet fever is much smaller than that of smallpox or of measles; that the continents of Asia and Africa, which, as we have seen, are among the chief seats of these two diseases and especially of smallpox, have been visited by scarlet fever at the utmost to a very small extent, allowing even for imperfections in the record. A further comparison of these infective diseases with regard *to the manner of their occurrence*, particularly a comparison of measles and scarla-

[1] 'Observ. médicales, &c.,' pp. 36, 42.

[2] L. c., p. 208.

[3] See Mantegazza, 'Lettere med. sulla America meridionale,' Milano, 1856, i, p. 12; Dupont, 'Observ. sur la côte orientale d'Amérique,' Montp., 1868, p. 14; Rendu, 'Études topogr. et méd. sur le Brésil,' Paris, 1848, p. 66.

[4] According to the accounts by Piderit (in the 'Deutsche Klin.,' 1855, No. 16) and Gilliss ('U.S. Naval Astronom. Exped. to the Southern Hemisph.,' Washington, 1855, p. 247) scarlet fever was prevalent there first in 1829, or two years before its outbreak in Buenos Ayres; so that there may have been an importation from Chili to the east coast.

[5] 'Oest. med. Wochenschrift,' 1840, pp. 470, 697.

[6] 'Edin. Med. and Surg. Journ.,' 1840, April, p. 335.

tina, brings out some other interesting points of difference. It is especially significant that scarlet fever occurs *as an epidemic* much more rarely than measles, so that, at a given place, there may often be ten or twenty years or more between two successive epidemics of the former.

Thus, to give only a few particularly striking examples, there was at Upsala in the latter half of last century only one epidemic during a period of thirty-three years;[1] in Samara, as we have seen, the disease occurs so seldom that it is hardly known to the public; when the great epidemic broke out in Sweden in 1856, there were many places in Westerbottenslän which had been free from the disease for sixteen years;[2] in his account of the epidemic of 1870 at Bristol, Davies[3] states that many years had passed since the disease last showed itself there; Miquel[4] emphasises the infrequent occurrence of scarlatina in the department of Indre-et-Loire, many years often elapsing before an epidemic develops, and Meynet[5] makes the same statement for Lyons; Tourtual[6] gives it as a well-established fact that previous to the epidemic of 1822-3 at Münster, fifty years had elapsed without the disease occurring there; the last epidemic at Emden previous to 1839-40, had been in 1825-6, and there had been no scarlet fever for many years previous to that;[7] at Ulm for the seventeen years from 1838 to 1855 there was only one small epidemic;[8] before the epidemic of 1862-63 at Tuttlingen, the malady had not been seen for thirty-five years;[9] in Erlangen,[10] it had been epidemic three times in the forty years from 1819 to 1858; Urach was free from the epidemic for sixteen years from 1829 to 1844,[11] and there was a corresponding clear interval at Stuttgart from 1830 to

[1] Rosenstein, 'Kinderkrankheiten,' p. 417.
[2] Account in 'Sundheds-Collegii Berättelse' for 1856, p. 23.
[3] 'Brit. Med. Journ.,' 1870, Sept., p. 297.
[4] 'Gaz. méd. de Paris,' 1834, No. 27, p. 426.
[5] 'Lyon médical,' 1878, No. 49, p. 504.
[6] 'Hufeland's Journ. der pract. Heilkd.,' 1826, Dec. 3.
[7] Laporte, in 'Hannov. Annalen für die ges. Heilkd.,' 1841, n. s., i, p. 157.
[8] Majer, in 'Württb. med. Correspdzbl.,' 1857, p. 105.
[9] Heim, ib., 1864, p. 195.
[10] Küttlinger, in 'Bayr. ärztl. Intelligenzblatt,' 1860, p. 29.
[11] Rösch, in '[Bad.] med. Annal.,' 1843, ix, p. 561.

1846;[1] when the disease was prevalent in Baden in 1853-54, many places were attacked in which scarlatina had not been seen for fourteen years;[2] in Washington County, Ohio, there had been no epidemic for twenty-three years;[3] Boston from 1811 to 1830 was so little visited by scarlet fever that the cases of death from it in those twenty years numbered only forty.[4]

It is at the same time worthy of note that, if the disease once grow to an epidemic, it continues not unfrequently for several years in one degree or another, and often becomes diffused over wide stretches of country. Thus in 1825-26 it extended over Denmark, England, Germany, and France; in 1832-35 again over those countries together with Ireland and Russia; in 1846-49 over Germany, Denmark, England, and Scotland; in 1821 and 1851 over North America; in 1831-37 over the eastern countries of South America. The recurrence of these epidemical cycles is not associated, however, with any definite *periodicity;* and there is equally little regularity to be discovered in the return of the disease to particular localities. The scanty data that have been brought forward in support of a periodicity of that kind, are met by a much larger series of facts at variance with it; and the data in question lose all significance as the conviction grows, on analysing them, that some of the items necessary to complete the periodical series do not relate to actual epidemics, but only to aggregations of sporadic cases.

We have here another thing peculiar to the manner of appearing of scarlatina, which distinguishes it from measles; the latter occurs almost solely as an epidemic, the isolated cases of measles being either its forerunners or its scattered offshoots persisting for a time; whereas *sporadic cases of scarlatinal sickness,* in large numbers or small, have been observed a good many times.

A third conspicuous peculiarity of scarlet fever is the *variation in the intensity of the disease,* as seen in the degree of mortality, which in some epidemics is almost *nil,* or from three to five per cent. of the sick, and in others thirty

[1] Köstlin, in 'Archiv des Vereins für wissensch. Heilkde.,' 1865, p. 328.
[2] Account in the 'Mittheil. des bad. ärztl. Vereins,' 1855, No. 9.
[3] Hildreth, in 'Amer. Journ. of Med. Sc.,' 1830, Feb., p. 329.
[4] Shattuck, ib., 1841, April, p. 373.

per cent. and more. This varying degree of severity in the disease is shown, moreover, not only in the successive outbreaks following close upon one another at a given place, but also in the series of epidemics distributed over longer periods of time; it comes out, too, in an equally striking way on comparing the intensity of the disease in the contemporaneous outbreaks of localities adjoining, or even throughout a wide area.

"In the year 1801," says Graves,[1] "scarlet fever committed great ravages in Dublin, and continued its destructive progress during the spring of 1802. It ceased in summer, but returned at intervals during the years 1803-4, when the disease changed its character; and although scarlatina epidemics recurred very frequently during the next twenty-seven years, yet it was always in the simple or mild form, so that I have known an instance where not a single death occurred among eighty boys attacked in a public institution. . . . The long continuance of the period during which the character of scarlet fever was either so mild as to require little care, or so purely inflammatory as to yield readily to the judicious employment of an antiphlogistic treatment, led many to believe that the fatality of the former epidemic was chiefly, if not altogether, owing to the erroneous method of cure then resorted to by the physicians of Dublin, who counted among their number not a few disciples of the Brunonian school. . . . The experience derived from the present epidemic [1834-35] has completely refuted this reasoning, and has proved that, in spite of our boasted improvements, we have not been more successful in 1834-35 than were our predecessors in 1801-2." In a review of Armstrong's 'Practical Illustrations on the Scarlet Fever' a Boston critic observes:[2] "We will take this occasion to remark that not only during the last year (1819), but, with the exception of 1802, for more than thirty years past, the scarlet fever has not appeared under a severe form in this place. But the same has not been true in respect to some other parts of the country during the period above mentioned, and at former times this disease has been among the most

[1] 'A System of Clinical Medicine,' Dublin, 1843, p. 493.

[2] 'New England Journ. of Med.,' 1820, July, p. 253.

fatal scourges of New England." Drake speaks to the same effect of the disease as it occurs in the central (Mississippi) valley of the United States.[1] The history affords very many examples of great differences as regards benignity or virulence in the epidemics of scarlatina coexisting at several localities in the same neighbourhood. To refer to only a few of the more recent facts, we find evidence of this in the observations recorded of the epidemics of 1852 and following years in Würtemberg,[2] of 1853 in the Palatinate,[3] of 1855 in Middle Franconia[4] and Upper Bavaria,[5] of 1855 and 1856 in Sweden, and of 1857 in Pennsylvania.[6]

§ 53. Influence of Seasons and Weather.

The fact that by far the largest portions of Asia and Africa, particularly their tropical and sub-tropical regions, have almost entirely escaped scarlet fever up to the present, has often been taken as warranting the conclusion that *climatic influences* play a prominent part among those external factors which determine the geographical distribution of the disease. This opinion is entirely refuted by the proofs that scarlet fever has often been found epidemic in the tropical countries of South America; and if it cannot be denied, as Drake[7] first pointed out, and after him Minor[8] and Sozinsky,[9] that the States of the Union situated south of 33° have enjoyed a certain immunity in comparison with the Northern States, yet the reasons for that, as well as for the great rarity of the disease in the above-named regions of the Old World, are not to be sought, or at least exclusively sought, in circumstances of climate, the less so that in many

[1] L. c., ii, p. 596.
[2] Account in 'Württemb. med. Correspondenzbl.,' 1854, No. 26, p. 201.
[3] Account in 'Bayr. ärztl. Intelligenzbl.,' 1854, p. 434.
[4] Eckart, ib., 1856, p. 450.
[5] Wibmer, ib., 1858, p. 327.
[6] 'Transact. of the Pennsylv. State Med. Soc.,' 1858.
[7] L. c., ii, p. 596.
[8] 'Scarlatina Statistics of the United States,' Cincinnati, 1875.
[9] 'Philad. Med. and Surg. Reporter,' 1880, Jan., p. 69.

countries in cold or temperate latitudes, scarlet fever counts, as we have seen, among the rarest of diseases.

And just as the diffusion of the disease in space does not depend upon climatic influences, so also does its time of prevalence remain uninfluenced by *the season and the weather* much more than in the case of smallpox or measles, if indeed it be so influenced at all. In the accounts before me of scarlatinal epidemics in temperate latitudes, I find 435 in which the time of prevalence, and the point of highest intensity, are given with some exactness. A tabular statement of them brings out the following result:

	Winter.	Winter and Spring.	Winter to Summer.	Spring.	Spring and Summer.	Spring to Autumn.	Summer.	Summer and Autumn.	Summer to Winter.	Autumn.	Autumn and Winter.	Autumn to Spring.	Total.
Scandinavia and Russia	8	6	...	7	9	7	5	9	...	12	6	3	72
Germany, Holland, and England . .	46	24	4	21	19	19	25	36	14	48	25	6	287
France, Italy, and Spain . . .	7	7	1	5	4	1	6	7	...	6	4	3	51
North America . .	5	3	...	5	1	...	4	1	1	...	3	2	25
	66	40	5	38	33	27	40	53	15	66	38	14	435

The disease has been epidemic, therefore, 178 times in winter, 157 times in spring, 173 times in summer, and 213 times in autumn; or in 100 epidemics, there have been 29·5 in autumn, 24·7 in winter, 24 in summer, and 21·8 in spring. This conclusion, that the maximum falls in autumn and the minimum in spring while the prevalence in summer and winter is about equal, is confirmed by a second series of statistical data showing the mortality from scarlet fever for the several seasons over a long series of years.

Table of Deaths from Scarlet Fever.

	Years.	Seasons.			
		Winter.	Spring.	Summer.	Autumn.
London . . .	1838—1853	12,586	10,961	13,972	17,768
Sweden . . .	1864—1873	2,869	2,569	2,777	3,415

The percentage of deaths, accordingly, was:

For London 32·1 in autumn, 25·2 in summer, 22·8 in winter, 19·9 in spring.

For Sweden 29·4 in autumn, 23·9 in summer, 24·6 in winter, 22·1 in spring.

The result agrees, further, with the statements of the great majority of observers; next to autumn they put summer and winter as the seasons of greatest prevalence, assigning a somewhat lower place to spring.[1]

It does not appear, at first sight, what may be the cause of these differences in the frequency of scarlatina during the several seasons of the year; one thing at least is clear, that we should not look for the reason in the influence of particular *kinds of weather*. If some observers conclude from their collective experience that cold weather, particularly when accompanied by moisture, is especially favorable to the production of scarlatina, it must be kept in mind, on the other hand, that the disease has not only been prevalent over and over again during great heat and drought, but also that the epidemics occurring under those circumstances have reached their height precisely as the temperature rose, and have declined when the heat subsided and cooler weather set in; and further, that by far the larger number of observers altogether deny that the weather has any influence at all upon the origin and diffusion of the disease.

As examples of epidemics during the strong heats of summer the following may be mentioned:—1759 at Lille,[2] 1769 at Rouen[3] and Paris,[4] 1763 in Cephalonia,[5] 1723 at Lyons,[6] 1800 at Paris,[7] Würzburg,[8] Lüneburg,[9] Plauen,[10] and

[1] Spring is given as the proper season of scarlet fever by only a few French and Italian observers, such as Simonin for Nancy ('Recherches topogr. et méd. sur Nancy,' Nancy, 1854, p. 281), Priou for Nantes ('Journ. gén. de méd.,' 1826, Sept., p. 350), and Menis for Brescia (l. c., p. 154).

[2] Boucher, in 'Journ. de méd.,' xi, p. 92.

[3] Lepecq, 'Samml. von Beobachtungen über epid. Krankheiten, &c.,' p. 290.

[4] Desessartz, in 'Journ. de méd.,' xlix, p. 538.

[5] Zulati, l. c.

[6] Gilibert, 'Adversar. med.-pract.,' Lugd., 1791, p. 184.

[7] Account in 'Journ. gén. de méd.,' ix, p. 182.

[8] Gutberlet, in 'Hufeland's Journ. der pract. Heilkd.,' 1806, xxiii, pt. i, p. 29.

[9] Fischer, ib., 1801, xiii, pt. 4, p. 23.

[10] Schmöger, ib., 1805, xxii, pt. 2, p. 122.

other places in Germany, 1814 in the Prachiner district,[1] 1819 at Zell,[2] 1822 at Prague,[3] 1830 at Pittsburg (Pa.),[4] 1834 at Amsterdam,[5] 1838 at Charleston (S. Car.),[6] and 1847 at Dorpat.[7] Epidemics coming to a height as the temperature rose, and subsiding when the cold weather set in, were observed in 1778 at Birmingham,[8] 1786 in London,[9] 1791 at Ciotat,[10] 1822 at Fulda,[11] 1828 at Buer,[12] 1830 at Annaberg,[13] 1833 in Paris,[14] and 1846 at Smolensk.[15]

Referring to epidemics in Denmark, Salomonsen has the following remark on the relation of the disease to the state of the weather:[16]—"For Epidemierne i dette Aarhundrede synes det altsaa at vaere Regel, at de kun viste sig hos os vid en Temperatur, der var over den saedvanlige. I de œvrige meteorologiske Forhold kunne vi derimod ikke finde noget constant."

§ 54. Unaffected by the Nature of the Soil.

There is no reason whatever to suppose that *conditions of the soil*, such as elevation, configuration, or geological and physical characters, have the slightest influence upon the occurrence and spread of scarlet fever. Like smallpox and measles, it has been found just as often in mountainous as in low-lying districts, in valleys as on tablelands and plains,

[1] Sazyma, in 'Oest. med. Jahrb.,' 1829, n. s., i, p. 134.
[2] Andreae, in 'Rhein. Jahrb. für Med.,' ii, pt. 2, p. 32.
[3] Bischoff, 'Darstell. der Heilungsmethode in der med. Klinik.,' &c., Prag., 1825, p. 26.
[4] Callaghan, in 'Amer. Journ. of Med. Sc.,' 1831, May, p. 71.
[5] Nieuwenhuys, in 'Transact. of the Prov. Med. Assoc.,' iv, p. 71.
[6] Logan, in 'Southern Med. Reports,' l. c.
[7] Jamson-Himmelstiern, in 'Rigaer med. Beiträge,' i, pt. 1, p. 144.
[8] Withering, 'Account of the Scarlet Fever, &c.,' Lond., 1779, p. 35.
[9] Sims, in 'Mem. of the Lond. Med. Soc.,' i, p. 388.
[10] Ramel, in 'Journ. de méd.,' lxxxviii, p. 169.
[11] Schwarz, in 'Rhein. Jahrb. für Med.,' xii, pt. 1, p. 75.
[12] Krebs, in 'Heidelb. klin. Annal.,' 1833, ix, p. 137.
[13] 'Physikats-Berichte aus dem Königreich Sachsen v. d. Jahren 1828-30,' p. 65.
[14] Account in 'Gaz. des hôpit.,' 1833, No. 129.
[15] Metsch, in 'Med. Ztg. Russl.,' 1846, p. 319.
[16] 'Udsigt over Kjöbenhavns Epidemier,' Kjöbenh., 1854, p. 38.

on the most various formations whether old or recent, on dry ground and on wet; and if some localities and tracts of country have been more severely visited than other districts near them at times when scarlet-fever epidemics have been general, yet the local circumstances in question give no clue one way or another.

§ 55. Mildness or Severity of the Type of Disease.

Scarlet fever shows itself to be independent of climate, season, weather, and influences of locality, not only as regards its distribution-area, but also as regards its intensity, or *the mild or malignant type of the epidemic*. Just as little, speaking generally, do *hygienic defects* arising from social conditions appear to exert any definite influence on the character of the sickness. In tracing the geographical area of the disease, I have already shown that in whatever tropical regions it is met with, just as in temperate latitudes, it prevails at one time mildly and at another time in a disastrous form; the epidemics on the East Coast of South America, in the West Indies, and in Peru are examples. The two following series of observations show how little the benign or malignant type of the epidemic is influenced by the season of the year.

Among the epidemiological data before me I find the season of prevalence and the type of the sickness given somewhat precisely for 265 epidemics. Of these 126 are given as of mild type, and 139 as severe;[1] in winter there occurred 77 (34 mild and 43 severe), in spring 50 (27 mild and 23 severe), in summer 66 (30 mild and 36 severe), and in autumn 72 (35 mild and 37 severe). The percentage, accordingly, is as follows:

Of 77 winter epidemics	44·2	per cent.	*mild*,	55·8	per cent. *severe*.
„ 50 spring	„ 54·0	„		46·0	„
„ 66 summer	„ 45·5	„		54·0	„
„ 72 autumn	„ 48·6	„		51·4	„

[1] In order to determine what constituted a "severe epidemic" I took either the express declaration of the writer or the rate of mortality, reckoning as severe all epidemics with a mortality of over 10 per cent.

Thus the maximum of malignancy falls in winter and summer, the minimum in autumn; but the difference is so small that it need hardly be considered.

In Sweden, for the years 1864-1873, the sickness and mortality from scarlatina were as follows:

In the winter months	out of	16,188	sick,	2869	died	=	17·7	per cent.
„ spring	„	„	14,716	„	2749	„	= 18·7	„
„ summer	„	„	13,997	„	2877	„	= 20·0	„
„ autumn	„	„	20·096	„	3415	„	= 17·0	„

Here the mortality is greatest in summer and spring, while the minimum falls in autumn; but there, too, the differences are so insignificant that no particular stress need be laid on them. We may therefore dismiss the notion, derived from isolated observations,[1] that epidemics of scarlatina are of an especially severe type in changeable and damp weather, an idea already refuted, as we have seen, by the occurrence of severe epidemics during the dry and hot weather of summer.

By other writers it has been sought to connect the severity of the epidemic with the low and damp situation of the affected locality, and with the injurious effects of products of decomposition in the soil or in stagnant water; and in this way an explanation has been furnished of the relative frequency of the disease in towns as compared with country districts. Thus, Cremen[2] would attribute the malignant character of the Cork epidemic of 1862 to the effects of putrid effluvia (from bad drains, dung-heaps, &c.), which were perceptible in those parts of the town where the poorer classes lived and to which the sickness was almost confined. Ballard[3] points out that in the epidemics of 1857-68 at Islington the streets situated near the watercourse suffered most; Andreae[4] adduces the fact that the town of Zell

[1] Morris ('Lectures on Scarlet Fever') for Philadelphia, Attenhofer ('Med. Topogr. von St. Petersburg,' Zürich, 1817, p. 240) for St. Petersburg, Thuessink (l. c.) for the Netherlands, Gutberlet ('Hufeland's Journ.,' l. c., p. 102), from observations at Würzburg, 1789-1803, and Wittmann ('Rhein. Jahrb. für Med.,' 1822, v, pt. 1, p. 59) from observations made in Mainz and neighbourhood, 1818-20.

[2] 'Dubl. Quart. Journ. of Med.,' 1863, May.

[3] 'Brit. Med. Journ.,' 1869, Dec., p. 620.

[4] L. c.

suffered more in the epidemic of 1819 than the neighbouring mountainous districts; Marchioli[1] observes that during the Cremona epidemic of 1871 the only place near, that was attacked, was the village of Ricorsano, occupying a marshy situation, all the elevated and dry localities escaping; Seifarth[2] remarks of the Langensalza epidemic of 1867, that the streets mostly affected were those near the churchyard where the soldiers that fell in the battle of 1866 were buried. Carpenter[3] gives several cases observed in and around Croydon from 1848 to 1870, which tell in favour of the injurious influence of putrid emanations on the course of the disease in particular individuals or in groups of persons. Murchison also,[6] while he admits that the fever had fewer victims in the densest and unhealthiest parts of London than in the cleanest and most sparsely populated, still thinks that some weight should be laid on the unfavorable influences which effluvia from drains exert on the course of the malady. We have also to consider here the circumstance already mentioned, that scarlet fever is not only of more frequent occurrence and more widely spread in towns where those sanitary defects are most felt, but also that it is of a more malignant type than among the country people,—that the great centres of traffic are the principal seats of the disease, and of the disease in its worst forms. Thus, Radcliffe[5] has shown that in England the mortality is greatest in London and in the north-western and northern counties (Cheshire, Lancashire, Yorkshire, Durham, Northumberland, Cumberland, and Westmoreland), the centres of mining and manufactures, while it is least in the southern and inland counties of Middlesex, Herts, Bucks, Oxford, Notts, Huntingdon, Bedford, and Cambridge, in which the population is more scattered.

In all these cases, as in many others of a like kind, it is clear that we have to deal with an aggregate of injurious influences, the pathological significance of each of which can hardly be estimated by itself. As a general principle we

[1] 'Gaz. med. Lombard.,' 1872, No. 14.

[2] 'Zeitschr. für Epidemiologie,' 1869, n. s., i, p. 56.

[3] 'Lancet,' 1871, Jan., p. 111.

[4] 'Lancet,' Oct. 15th, 1864.

[5] 'Transact. of the Epidemiol. Soc.,' 1867, ii, p. 265.

shall have to admit for scarlet fever, as for most other epidemic diseases, that whatever lowers the power of resistance in the individual, raises not only his predisposition to take the disease, but also the risk to which his life is exposed by it. We find this law brought out in the circumstances before named, and above all in those records[1] which prove the disease to have been severe and attended by high mortality in many epidemics among the proletariat, where want, misery, neglect, dirt, &c., along with defective or altogether deficient medical attendance, render the situation of the sick as unfavorable as it can be. But there cannot well be any question of attributing to these defects a *specific* influence upon the type and character of the epidemics as a whole. The arguments of English observers, who have always given special attention to the point, are most convincing in this respect. Thus Withering, one of the earliest and best authorities on scarlatinal epidemics, states, on the authority of his Birmingham experiences in 1778, that the disease had raged severely in many elevated, dry, and airy localities, while the dwellers in low, damp, and ill-ventilated parts of the town suffered to a very slight degree or not at all. Graves, in his account of the epidemic of 1839 in Ireland,[2] says: "The nature of the disease did not appear in the least connected with the situation or aspect of the patient's dwelling, for we observed it equally malignant in Rathmines as in Dublin, on the most elevated habitations on mountains as in the valley of the Liffey. It raged with similar violence at Kingstown and the neighbourhood of Killiney and Bray." Wood,[3] in his account of the epidemic of 1835 at Edinburgh testifies to the same effect: "It is a remarkable circumstance that the fever extended nearly as

[1] See the epidemiological accounts by Cohn (in 'Casper's Wochenschr. für die ges. Heilkde.,' 1833, p. 913) for Inowraclaw 1831, by the Medical Council of Rhenish Prussia ('Jahresbericht,' 1838, p. 42) for the department of Coblenz 1838, by Möller ('Archiv für physiol. Heilkd.,' 1847, vi, p. 535) for Königsberg 1844-45, by the Würtemberg physicians ('Württemb. med. Correspdzbl.,' 1854, No. 26, p. 201) in 1852, by Heim (ib., 1864, No. 25, p. 195) for Tuttlingen, 862-63, by Cremen (l. c.) for Cork same year, by Lievin ('Vierteljahrschrift für öffentl. Gesundheitspfl.,' 1871, iii, p. 369) for Danzig 1868, and by Otto ('Das Scharlachfieber in Chicago, &c.,' Göttingen, 1879, p. 19) for Chicago, 1876-77.

[2] 'A System of Clinical Medicine,' Dublin, 1843, p. 501.

[3] 'Edin. Med. and Surg. Journ.,' 1837, July, p. 99.

rapidly, proved as severe, and was as destructive of life in the families of the higher ranks, living in large houses, in open, airy situations, as in those of the lower, crowded together in small, airless dwellings, in narrow streets and lanes." In the report[1] upon the epidemic of 1868 in London, all the metropolitan Medical Officers of Health agree in stating that the well-to-do part of the population living under favorable conditions suffered as much as the poor, and sometimes more. The account of the epidemic there in 1869-70 is to the same purport: "The disease," says Whitmore in his report for Marylebone,[2] "was by no means especially prevalent amongst the dirtiest and most destitute classes; it principally occurred amongst children of the respectable poor, and more frequently than otherwise in houses where the sanitary conditions were by no means defective." In the report by Davies[3] of the epidemic of 1870-71 at Bristol it is stated: "Judging from the class of persons and houses mostly affected by the present epidemic, this disease has but little if any connection with general sanitary conditions. . . . It has proved its fatal influence as readily in the well-situated and well-ventilated mansions of our healthy and rich suburb of Clifton as in the crowded courts and badly-ventilated alleys of the more ancient parts of the city. Indeed, *the old and badly-ventilated courts enjoy a considerable immunity.*" Hillier[4] says: "This disease does not confine its ravages to the dwellings of the poor, nor does it commit much greater devastations in ill-drained, badly-ventilated places than in those which are well provided with drainage and moderately supplied with fresh air. Hygienic conditions exercise less influence over its course than they do over most epidemic disorders. In the great majority of deaths from measles there are generally either unfavorable sanitary arrangements surrounding the patient, or the child was previously unhealthy; deaths from measles, except in the case of unhealthy children, are rare amongst those in easy circumstances. The same cannot be

[1] 'Med. Times and Gaz.,' 1869, April, p. 367.
[2] 'Brit. Med. Journ.,' 1870, May, p. 526.
[3] Ib., 1870, Sept., p. 297.
[4] 'Med. Times and Gaz.,' 1862, 31st May.

said of deaths from scarlatina; it has even been asserted by some that this disease proves more fatal to the children of the rich and well-to-do classes than to those of the poor." Prior,[1] in his account of the epidemic at Bedford in 1855, says: "There is nothing in these facts in the shape of clear proof that either proximity to the river or geological formation influenced the extension or fatality of the disease." There, also, the well-to-do suffered equally with the poor; and so far as relates to the notion that defective drainage has an unfavorable influence on the course of the disease, Prior states that "there was nothing in this epidemic to sanction such a belief."

I think that the facts here adduced, to which I could add many more of a like kind, including my own observations at Danzig from 1846 to 1860, will suffice to show that we are completely in the dark as to the conditions that make scarlatinal epidemics to assume a good or bad type. Science at the present day has to confess ignorance on this point, just as Drake did when he said:[2] "of the causes or conditions which determine these remarkable diversities of phenomena and danger [in scarlatina] we are entirely ignorant."

§ 56. The Virus of Scarlatina.

Concerning the *nature of the scarlatinal poison*, its specific character is beyond all question, and like all other of the so-called morbid poisons, it may with good reason be taken to be an organised body. But all the inquiries and experiments hitherto—by Hallier,[3] Coze and Feltz,[4] Böning,[5] Balogh,[6] and others—have not furnished any certain knowledge about it. The well-founded presumption of the organic nature of the virus of scarlet fever excludes the idea of an autochthonous pathogenesis: the disease never arises but in conse-

[1] 'Lancet,' 1869, Oct., p. 570.

[2] 'Principal Diseases of the Interior Valley of North America,' Cincinnati, 1850, ii, p. 596.

[3] 'Jahrb. für Kinderheilkde.,' 1869, ii, p. 169.

[4] 'Gaz. méd. de Strasbourg,' 1869, Nos. 1—4.

[5] 'Deutsche Klinik.,' 1870, No. 30.

[6] 'Med. Central-Ztg.,' 1876, p. 625.

quence of a conveyance of the specific poison, and the spread of the sickness is dependent on the fact that the virus, reproduced in the sick person and eliminated by him, forms new foci of infection.

§ 57. Original Habitat Uncertain.

At the outset of this inquiry we found reason for believing that no conclusion could be arrived at as to *the native habitats of scarlet fever*,—from what points of the globe the disease had originally issued, or where it had subsequently become domesticated. So much only one may affirm, on the strength of the historical facts above given, that the endemic area of scarlet fever is a limited one, that at many parts of the globe the disease has never occurred except when imported, that it has been able, indeed, to exist in those countries for a longer or shorter period, but has died out in the end, and if it has reappeared it has always been necessary to assume a fresh introduction of the morbid poison. The reason why the continents of Asia and Africa have remained hitherto almost free from the sickness, notwithstanding many importations of it, does not admit of being explained; the influences of climate, soil, or other conditions perceptible to the senses, afford at least no clue; and just as little is the cause of the immunity to be looked for in the physiological characters of the natives of these regions, or, in other words, in *the peculiarities of race*. Pruner,[1] indeed, states that so far as he knows, the coloured races are quite exempt from scarlet fever, but that opinion is certainly wrong. Scarlatina occurs, although rarely, among the negro population of Senegambia;[2] and this infrequency is explained, not by any relative immunity of the negro race, but by the rarity of the disease there at all. Negroes in the United States suffer like the whites, and, it would seem, in almost equal proportion; according to Frick,[3] the proportion among whites and negroes in the Baltimore epidemic of

[1] L. c., p. 120.

[2] See Moulin, 'Pathologie de la race Nègre, &c.,' Paris, 1866, p. 22.

[3] 'Amer. Journ. of Med. Sc.,' 1855, Oct, p. 321.

1850-54 was 13·8 and 10·8 in 10,000 inhabitants. In South America, following the accounts of Brunel,[1] Sigaud,[2] and others, the disease is equally frequent and of the same type among the coloured as among the white races; Mantegazza,[3] indeed, states that the creoles suffer more than the whites; and, that the red-skinned natives of North America (Canada) are at least not exempt, follows from Stratton's remark;[4] "in epidemic scarlatina it appeared to me that the Indians were less susceptible of an attack than the whites."

§ 58. Diffusion of the Morbid Poison.

The *vehicles of the morbid poison*, conveying the malady from person to person and from place to place, have been taken to be the currents of air about the patient, or articles in that infected atmosphere which had taken up the virus eliminated from the sick. The distance to which the virus may be carried by the air cannot be expressed in definite figures; there is evidence at least that the distance is a small one, the focus of infective action being often closely circumscribed, and the immediate neighbourhood free. It remains an open question whether this fact, as it stands, is to be explained by a loss of potency in the poison through diffusion and dilution, or on the hypothesis that the virus is a relatively heavy body which can be carried only a short distance by the atmospheric currents. There are many instances truly classical, going to prove *the conveyance of the poison through the medium of infected articles*. Thus, to give only a few of the more recent observations on this point, Heslop[5] traces the spread of scarlatina in the Birmingham Children's Hospital to the linen of children sick with scarlet fever being aired and made up in the same room with the rest of the house-linen; formerly the precaution had been taken to have the body- and bed-linen of scarlatinal patients washed outside the institution, and so long as that rule was

[1] L. c., p. 42.
[2] L. c., p. 209.
[3] 'Lettre, &c.,' i, p. 12.
[4] 'Edin. Med. and Surg. Journ.,' 1849, April, p. 282.
[5] 'Lancet,' 1870, Nov., p. 736.

followed, there had never been any conveyance of the disease. Ogle[1] also mentions similar cases where the disease had been communicated by linen from the laundry. He refers besides to a case which shows that the virus may cling to furniture. A husband and wife, having lost four children by scarlatina, removed for a time to another locality with their only surviving child; they had the rooms that had been occupied by the deceased children thoroughly cleaned, the walls re-papered, all clothes and bed-linen that had been used by the sick destroyed, and the furniture disinfected; only two arm-chairs, of which the hair stuffing was torn, were overlooked in the cleansing operations. Ten weeks afterwards, the family returned to their house, and in fourteen days the child sickened of scarlatina. Petersen[2] mentions an attack of scarlet fever in a young girl, who kept up a correspondence with a friend at a distance; the latter had been suffering from scarlet fever, and several of her letters had been despatched during the period of desquamation. Many other examples of the disease being carried by letters are to be found in English writings; and in none of those cases was there any other way of explaining the origin of the disease.

Since the attention of the profession has been drawn to the communication of infective diseases by drinking water and milk, observations have not been wanting in support of that hypothesis for scarlet fever; but whether they are conclusive or not is open to question. Thus Pride[3] calls attention to the fact that in the epidemic of 1868 at Neilston, those attacked were mostly children who lived in certain houses drawing their water supply from polluted wells. Bell Taylor,[4] Welch,[5] Buchanan,[6] and others give particulars of scarlatina breaking out under circumstances which made it probable that the infection was produced by milk supplied by milk-dealers in whose families there had been cases of scarlatina.

[1] Ib., Dec., p. 881.

[2] 'Ugeskrift for Laeger,' 1871, iv, p. 309.

[3] 'Glasgow Med. Journ.,' 1869, p. 440.

[4] 'Brit. Med. Journ.,' 1870, Nov., p. 489.

[5] Ib., 1876, Aug., p. 225.

Report of Medical Officer of Privy Council, &c.,' 1875, n. s., No. vii, p. 72.

Finally, as regards the often observed conveyance of scarlet fever by individuals who have themselves remained well, that is explained without difficulty on the assumption that their persons or clothes had become carriers of the morbid poison. The history of scarlatina abounds in examples of that kind, and it has generally fallen to the lot of medical practitioners to be the bearers of misfortune in these cases; I can myself recall a case in my own experience where a physician brought the infection to his wife during her lying in.

CHAPTER VII.

MALARIAL DISEASES.

§ 59. Malarial diseases form a nosological group so closely related to one another both in their pathology and therapeutics, and also from the point of view of their history and geography, that we are entitled to regard them as modifications of a single morbid process; and we may apply to that process a name which commits us, at least, to no theory, namely, *the malarial process.*

Not many of the forms of people's sickness can be followed with so certain a clue as the malarial, through all intermediate periods up to the first beginnings of scientific medicine, although the ancient and mediæval chronicles, medical and other, do not enable us to estimate the extent of their epidemic and endemic prevalence. It is, indeed, with the epidemiological records of the sixteenth century that the historical research begins; and, for the geographical distribution of malaria, it is on the observations published since the commencement of the present era that we have practically to depend. Following up each of those lines of inquiry, we come to recognise in the malarial process a form of disease which hardly any other of the acute infective diseases can compare with in respect of its area of distribution over the earth. Covering a broad zone on both sides of the equator, the malarial diseases reach their maximum of frequency in tropical and subtropical regions. They continue to be endemic for some distance into the temperate zone, with diminishing severity and frequency towards the higher latitudes; in epidemic form they not infrequently appear in yet other regions; and, in still wider diffusion with the character of a pandemic, also beyond their indigenous latitudes.

The geographical distribution of the malarial diseases stands, therefore, in a certain definite relation to the latitude; and, in correspondence with that principle of diffusion, we shall find the most suitable course of investigation to be one proceeding from the equator to the poles.

§ 60. Geographical Distribution.

Africa.—First and foremost, we meet with one of the most intense malarial regions of the eastern hemisphere in *the tropical part of the continent of Africa* and the islands adjoining thereto. In the *basins of the Senegal*[1] *and Gambia*[2] the disease is enormously frequent and malignant, equally on the marshy coasts and river banks and in the relatively dry regions of the upper river basin. Next, over the whole *Guinea Coast*[3] from Sierra Leone down to Cape Lopez, but more especially in the basins of the Niger[4] (Benin and the Slave Coast) and the Gaboon;[5] and, further, on the coast of Sierra Leone,[6] the Ivory[7] and Gold[8] Coasts, and in the adjoining islands of Fernando Po[9] and St. Thomas.[10]

For an approximate estimate of the frequency of the disease in these regions, the fact may suffice that among 15,469 negro troops located in the three English stations of Gambia, Sierra Leone, and the Gold Coast (including Lagos) during 1859-75, there occurred 4983 cases of malarial fever; or, in other words, 32 per cent. of the troops (native)

[1] Thevenot, p. 258, Berville, Leblanc, Dutroulau, p. 248, Beal, Borius (I), Mondot, p. 12, Gauthier, pp. 16, 18, Chassaniol, Barthélemy-Benoit, Mahé, Chabbert, Thaly, Bourse (I), Serez, Berenger-Féraud, Léonard, Carbonnel, Verdier, Dudon, Defaut. (The particulars of these and all subsequent references will be found in the syllabus of the literature at the end of the section).

[2] Roe, Ritchie (April), p. 323, Rey (I).

[3] Bryson, Kehoe, Daniell (II), p. 154, Ritchie (April), p. 324 (May), p. 402 (June), p. 515, Heyne, Lawson.

[4] Isoard, Oldfield, McWilliam, p. 180, Pritchett, Quintin, Féris (I).

[5] Touchard, Griffon de Belny, Monnerot, p. 14, Bourse, p. 38, Rouvier, Abelin.

[6] Boyle (I), p. 123, Clarke (I), Gore.

[7] Legrain, Jubelain, Guergueil, Forné, Michel.

[8] Gordon (I), Lucke, Gardiner in 'Brit. Army Med. Reports,' v, p. 323, Moriarty, Michel.

[9] Daniell (II), p. 154.

[10] Id. (II), p. 189.

suffered from that disease. The amount of sickness among the European population was naturally much greater.

There is better health from Capo Lopez downwards, and along the *Congo Coast*,[1] where the regions of more intense malaria are met with only at intervals, as in the swampy neighbourhood of Benguela;[2] and this exemption from malarial disease becomes more and more marked the nearer we approach the *Cape of Good Hope*,[3] which itself enjoys, along with *St. Helena*, an almost complete immunity from the endemic fever. The same exemption obtained, up to a few years ago, in the islands of Réunion[4] and Mauritius,[5] situated within the tropics; but since 1866, centres of intense malaria have developed in these colonies under the circumstances to be afterwards mentioned.

Among a total of 84,814 English troops maintained at the Cape during 1818-36, there occurred 1632 cases of malarial fever, a rate of sickness amounting to 1·9 per cent.; but by far the larger part of all these cases were troops from India or China (and in 1867-75 from Mauritius) in whom the fever had recurred; in the years when there was no such influx, the number of cases was exceedingly small. At St. Helena from 1859 to 1869, seven cases of intermittent fever were observed among 5462 English troops. In the Mauritius, the number of admissions for malarial fevers in 1812-36 and 1859-66 amounted to 262, or 0·4 per cent., and there also the disease occurred almost exclusively among such of the troops as had been transferred from China or India shortly before; but in the period from 1867 to 1875, or after the disease had developed in the island, 6970 admissions occurred among 6084 men, and of these there occurred, in the first three years (1867-69), 5048 admissions among 3201 men. Of this frightful epidemic, further particulars in the sequel.

A second great malarial region of the African continent is formed by the *East Coast* from Delagoa[6] Bay upwards along the littoral of *Sofala*, *Mozambique*,[7] and *Zanzibar*;[8] the trustworthy accounts of travellers[9] place it beyond doubt that the

[1] Moreira, Tams, Bastos, Ritchie, (May) p. 411, Magyar (I), 450, Hugiot.
[2] Falkenstein, in his account of the Loango Coast.
[3] Friedel (II), p. 112, Egan.
[4] Barat, Bassignot, Lacaze.
[5] Allan, Tessier, Monestier, Rogers, Labonté.
[6] Allan, Boteler.
[7] Roquete.
[8] De la Peyre, Ruschenberger, Semanne, Lostalot-Bachoué.
[9] Livingstone, Miller, Fritsch.

foci of malaria there extend far into the interior, from the shores of the Zambesi, Schiré, and Rovuma, and beyond Lake Ngami to the northern border of the Kalahari desert. Not less common and pernicious is the disease in the *Comoros*[1] and in *Madagascar*,[2] where the north-east coast only, and the mountainous part of the interior, enjoy more favorable conditions of health. The extensive plateau of *Somali Land*, owing to its generally elevated position, the dryness of the soil, the absence of swamps on its thinly wooded coast, and the circumstances of its climate, probably deserves the character for comparative healthiness which certain travellers have given it. Also in those parts of *Abyssinia*[3] that are subject to the same influences, malarial diseases occur only to a moderate extent and of a relatively benign type; this holds good for the strip of coast (mostly narrow), with the exception of a few marshy points such as Massowah (which is almost uninhabitable for Europeans on account of its malaria),[4] as well as for the whole *west coast of the Red Sea* generally,[5] and it holds good also for the Abyssinian highlands; while malarial diseases are endemic in their most pernicious forms in the narrow, densely-wooded and damp river valleys, in the swampy flats of Seraë, Lower Samen, &c., as well as on the shores of the Takazzé and of Lake Zuaie.

The countries of which we have just been speaking form the eastern portion of a third great malarial region, which extends from the western slopes of the Abyssinian highlands, across *Nubia*,[6] and a great part of the Soudan (as much of it as is known), and through the marshy flats of *Kordofan* and *Darfur* as far as Lake Tchad (whose shores are the seat of the worst kind of fever), and probably beyond it as far as the elevated plains to the west. In Nubia the chief endemic seats are the banks of the two arms of the Nile, more espe-

[1] Curtis, p. 26, Dutroulau, p. 42, Grenet, Foncervines.

[2] Rochard, Daullé, Dutroulau, p. 239, Panon de Fagmoreau, Chabbert, Vinson (I), Davidson, Borius (III), Borchgrevink.

[3] Aubert-Roche, Harris (I), iii, 165, Pruner, p. 356, Courbon, pp. 15—30, Martin.

[4] Blanc.

[5] Aubert-Roche, Coulson.

[6] Veit, Pruner, p. 356, Griesinger, Hartmann (I).

cially *Khartoum* situated at their confluence, and the Nile valley from that point down to Dongola. Then comes a region free from malaria, including the northern parts of the Dongola steppe and the rocky plateaus of that country as well as *Upper Egypt*, and the greater part of *Middle Egypt*;[1] that again is succeeded by a malarial zone which includes the low-lying province of *Fayoum* in direct connexion with the Nile valley, and follows the river from Cairo to the shores of the Mediterranean, becoming broader as it advances northwards, and extending more particularly over as much of *Lower Egypt* as is watered by the Nile.

In *Tripoli* the basin-shaped province of *Fezzan*, abounding in salt lakes, together with the oases, is stated to be the seat of pernicious endemic malarial diseases; in *Tunis*[2] also they are prevalent under the same circumstances. Finally, we meet with a very extensive region of malaria on African soil in *Algiers*.[3] The coast zone is here the headquarters of the disease. Among particular coast localities there are: in the Province of Constantine,—Bona, Philippeville, and Gigelhy; in the Province of Alger,—the plain of Metidja, Alger, Blidah, Koloah and Tenes; in the Province of Oran,—Mostaganem, Oran, Ain-Temouchen, and others. But malarial diseases are also widely spread on the uplands of the Greater and Lesser Atlas, on the banks of the Seybus, in Constantine, Setif, Batna (Province of Constantine),[4] in Medeah, Milianah, Teniet-el-Had (Alger),[5] Tiaret, Mascara, Sidi-bel-Abbes, Saida, Tlemcen (Province of Oran),[6] and in many oases of the Great Desert, such as Biskara, Tuggurt,[7] Ouaregla,[8] and Lagouat.[9] From those disease-centres, we pass to the great malarial region of the Soudan. According to an approximate calculation, the yearly number of admis-

[1] Ib.

[2] Ferrini, p. 201.

[3] Maillot, p. 265, Périer, Furnari, Laveran (I), Langg, Jacquot (I), Espanet, Beaunez, Bertherand (I, II), Catteloup (I), Haspel (II), p. 151, Armand (I), p. 64, Philippe, Leclerc, Bertrand, Gaucher (I, II).

[4] Worms, Guyon (I), Antonini (I, II), Goudineau, Corne, Bedié, Quesnoy, Mouchet.

[5] Villette, Bertherand (III), Finot, Durand, Laveran (II), Seriziat, Claudot.

[6] Guyon (II), Marseillhan, Cambay, Froussart, Sourier et Jacquot.

[7] Audet. [8] Creissel. [9] Bachon.

sions for malarial diseases among the French troops in Algiers amounts to about one half of the admissions for all diseases whatsoever occurring among them.

Asia.—We come next to the malarial regions of the tropical and sub-tropical parts of the *Asiatic continent* and the islands belonging thereto. Among these *Arabia*[1] takes a prominent place. In contrast to the western shore of the Red Sea, which is little infested by malaria, there is a region of very considerable malaria in the flat marshy strip of coast of the Hedjah (especially at Jeddah and Yembo),[2] and in Yemen, from Jisan downwards to Mocha. Aden, situated at the southern extremity of this coast, is free from endemic malarial fever ;[3] and, from the accounts of travellers, the sandy plateau of the interior should also enjoy favorable sanitary conditions. On the other hand, the disease prevails in its worst forms on the south coast, especially in Muscat,[4] along the marshy shores of the Persian Gulf and the adjoining islands (as at Bassadur in the island of Kishin[5]), as well as in a wide-spread endemic in the valleys of the Euphrates and Tigris from their mouths upwards throughout *Mesopotamia*.[6]

In *Syria*[7] we meet with extensive regions of malaria in the damp valleys of the Lebanon (equally in the valley of Beka, situated at a height of 1200 metres, and in the valley of the Jordan near the Dead Sea) ; further, at Jerusalem,[8] Damascus, Aleppo,[9] and other inland places, but especially in the coast localities, in Gaza, and up the coast to Jaffa, Tyre, Sidon, Beyrout,[10] Tripolis, Acre, and Skanderoum.

[1] Aubert-Roche, Pruner, p. 356, Palgrave.

[2] Courbon, p. 67.

[3] Howison, Courbon, p. 59. From 1863 to 1868 and from 1871 to 1872 there occurred among a total of 5219 English troops at Aden, 374 admissions for malarial fever; at least 100 of these have to be deducted as belonging to a regiment moved from Bombay, so that the rate of malarial sickness in Aden itself amounted to about 5 per cent. of the garrison.

[4] Lockwood, Evatt.

[5] Rozario.

[6] Floyd, Wagner (I), Hyslop.

[7] Pruner, p. 356, Richardson (I), Rafalowitsch, Rigler (II), p. 376, Wertabet.

[8] Tobler, p. 32, London.

[9] Guys, p. 63.

[10] Yates.

From these centres the malarial region extends to the soil of *Asia Minor*,[1] from Adana and Tarsus along its south and west coasts (including Smyrna),[2] the marshy banks of the Scamander, the plain of Troy,[3] and along the coast of the Black Sea, from Sinope, around the Gulf of Iskimio and Broussa to the Dardanelles. The accounts of travellers are too slight to enable us to say how far inland the disease is endemic.

The table land of *Armenia*,[4] with the exception of a few points, and the central mountainous region of *Transcaucasia* (Grusia)[5] are little subject to endemic malaria; on the other hand, it is prevalent to a great extent on the marshy steppe of the Kuban, in the damp valleys opening towards the Black Sea, on the banks of the Terek, especially in the neighbourhood of Kisljar, at low-lying places in Dagestan, but in its very worst forms in the valleys of Abchasia, Mingrelia, Guria, and Imeretia, in the valley of Alasan, on the Mugan steppe, on the banks of the Kura as far as Lenkoran, as well as in the plain watered by the Araxes. From that point the malarial region extends along the marshy shores of the Caspian to *Persia*,[6] where endemic malaria is met with in the provinces of Ghilan and Mazenderan (Asterabad), in the valleys of the Attrek and Gurgan opening towards Turkoman territory, at several points on the plateau of Teheran, but of the most pernicious kind on the shores of the Persian Gulf, especially in Bushire.[7]

Among the more considerable malarial regions of the continent of Asia we have to include *Beloochistan* and *Afghanistan*;[8] the endemic fever is met with in these countries equally on the swampy coast margins, as in Seistan abounding in salt marshes, in the lofty and dry Kelat, in the marshy plain of Dedar, and in the Bolan and Kandge Passes

[1] Black (II), Roeser, p. 31, Thirk, Pruner, p. 356, Rigler (II), p. 376, West.
[2] Clarke (II).
[3] Virchow (II).
[4] Wagner (I).
[5] Hirtzius, Mironow, Tschetyrkin, Reinhardt (I), Kaputschinski, Popoff, Moroschkin, Krebel, Liebau.
[6] Bell, Polack.
[7] Miller, Evatt.
[8] Hunter (I), Thornton, Harthill, Cook.

branching from it, and, further, in Kandahar and in the mountain valleys of Cabul and Jelalabad. The last of these joins on to the great malarial region which extends over the northern plain of Hindostan, from the Punjaub through Sind and part of the Bombay Presidency, the North-West Provinces and Bengal, corresponding respectively to the basins of the Indus and Ganges.

To estimate the amount of malarial disease in the several great territorial divisions of India, the following statistics, gathered from the returns of sickness among the British troops (Europeans) quartered in them, will afford a sufficient basis :[1]

Table of Admissions for Malarial Fever among European Troops in India.

Presidency.	Years.	Total number of Troops.	Admissions for Malarial Fevers.	Per-centage.
Bengal (including North West Provinces) . .	1847—1854[2] and 1860—1875	752,419	350,279	46·6
Bombay . . .	1860—1875	176,706	81,123	45·9
Madras . . .	1860—1875	181,695	25,686	14·1
		1,110,820	457,088	41·1

While, therefore, the average sickness from malarial fevers among European troops in India amounts to 41 per cent., it is as high as 46·6 in Bengal and the North-West Provinces, and as low as 14·1 in the Madras Presidency, and it almost reaches the former figure in the Presidency of Bombay. Calculated on the total admissions for all forms of sickness, malarial fevers amount to 61 per cent. in the Bombay Presidency, 55 per cent. in Bengal and the North-West Provinces, and 35 per cent. in the Presidency of Madras.[3]

[1] The tables are compiled from the 'Army Medical Reports,' published annually by the English Government; in making use of these reports I have exercised the greatest caution, and have disregarded the earlier of them, which are less reliable.

[2] Compiled by Macpherson, in the 'Indian Annals of Med.,' 1858, Jan., ix, p. 227.

[3] Day, ib., 1859, Jan., xi, p. 72. For the distribution of malarial diseases

This enormous amount of malarial disease in the North-West Provinces[1] and in the Presidencies of Bengal and Bombay is chiefly attributable to the endemic prevalence of the disease in the basin of the Indus, where the disease is met with over a vast area in highly pernicious forms, both in the upper part of the river system, *Peshawur*,[2] *Cashmere*, and the *Punjaub*,[3] and in the State of Sind[4] belonging to the lower basin. The disease occurs to a moderate extent on the dry sandy soil of Cutch,[5] while a region of intense malaria is formed by the jungle-covered and swampy plain of Guzerát.[6] Proceeding eastwards we find malaria endemic on the hilly plain of Maiwar,[7] which is free from marshes, and in Malwa; while it is in general more rarely met with on the barren sandy soil of the Rajpoot States. A focus of very intense malaria is met with, again, in the swampy slope which extends, under the name of the *Terai*, along the southern base of the Himalaya, through the provinces of Garhwal and Kumaon, and the State of Nepaul, to the borders of *Lower Bengal*[8] and far into the valley of the Ganges. In the upper regions of the Gangetic valley itself, such as the districts of Rohilkund, Allahabad, Sirhind, and Oude, the endemic foci of malaria are less frequent,[9] although severe epidemics of malarial fever have often occurred. On the Ganges the malarial region proper begins at Benares, where the flat river-banks are exposed to annual inundations, and it extends thence through the eastern part of the North-West Provinces (the fruitful valley of Tirhoot[10]), and through Lower Bengal,[11] where, next to the delta formed by the Hoogly[12] and Ganges, the chief seats of the disease are the marshy province of *Orissa*,[13] the richly-watered plain (marshy

throughout India in general see Henderson (I), Grierson, Annesley, pp. 33, 513, Morehead (I), p. 20, and Milroy.

[1] Murray (III). [2] Kinnis. [3] Moorcroft.

[4] Lord, Don, Inglis (II), Campbell, Collier, Kinnis.

[5] Winchester, Moore, Kinnis.

[6] Gibson (I), Inglis (I), Jackson (I).

[7] Ewart. [8] Curran.

[9] McGregor, pp. 15, 245, Jackson (II).

[10] Evans (I), Tytler.

[11] Martin, Twining (II), p. 250, Macpherson (I, II), Fleming, 'Army Med. Rep.,' 1861, p. 216, Forbes (I), Goodeve (I).

[12] Sutherland. [13] Sterling, Shortt (I).

in its northern parts) which descends between the Ganges and Burhampootra from the slopes of the Himalaya to the coast, and the flat swampy banks of the Burhampootra in the upper part of its course where it traverses the State of *Assam*.[1]

A point of especial interest for the history of malarial disease in India is raised by its endemic occurrence on the *table-land of the Deccan*.[2] These fevers occur even in the mountainous countries of Chota Nagpore and Gondwana, sloping on the east towards Orissa and on the west joining on to the Vindhya mountains, and there forming the boundary between the Deccan and the plain of Hindostan; and it is not only in the alluvial valleys, covered with jungle vegetation, or in the swampy valleys, that the fevers occur, but also, under the name of "hill-fever,"[3] on the absolutely dry soil of elevated points. It is those very hill-fevers that make up the greater part of the endemic malaria of the *Madras Presidency*.[4] The coast belt of that Presidency, perfectly flat, and for the most part sandy and scantily watered, is the part least affected, the disease being met with only at several scattered points, Masulipatam,[5] Nellore, Madras,[6] Pondicherry,[7] Tranquebar,[8] and other places on the Coromandel coast, where artificial irrigation, canals, or jungle plantations exert an influence special to the locality. It is the mountain region that here forms the proper habitat of the malarial diseases, particularly the hilly zone of the *Northern Circars*,[9] and the table-lands of *Bellary* and *Mysore*,[10] with the notorious fever-bed of Seringapatam.[11] In the southern division of the Pesidency the habitat of malaria is in the deep valleys, damp and covered with rice-fields or jungle, in the districts of Trichinopoly, Dindigul, Madura and Palamacotta, beyond which the endemic foci of malaria stretch far up into the hills. The *Malabar Coast* and all

[1] Leslie, McCosh.
[2] Staples, Hannah.
[3] Breton (I), Dunbar, Goodeve (II), Hughes and Anderson, Godfrey.
[4] Geddes (II), p. 87 ff, Balfour, Day (III), p. 74.
[5] Murray (I), Geddes, l. c.
[6] Cornish (July), p. 83, Shortt.
[7] Huillet.
[8] Ruhde.
[9] Wright (I), Macdonnell, Heyne, Smith (I).
[10] Eyre, Day (I), 1856, April, p. 571; 1858, Jan., p. 55.
[11] Nicoll, Geddes (I).

the western littoral belonging to the *Bombay Presidency*[1] is much more unhealthy than the east coast. Here also there is only a narrow margin of plain, but it is richly watered, abounding in woods and brackish lakes, marshy in part, or subject to periodical inundations, and therefore extensively malarious except at the more elevated and dry localities, such as Cananore, Tellicherry, and Calicut. Among the most important malarious regions of this zone are *Mangalore*, and the broad littoral territory of *Candeish*[2] at the mouth of the Tapti; but the disease is met with besides among the hills and valleys of the *Western Ghâts*,[3] from Balgâm[4] upwards through the territories of Savant-Warri,[5] Kolapore,[6] Rutnagherry,[7] Sattara,[8] and Poona,[9] to Ahmednuggar and Aurungabad.[10]

Among Asiatic countries in which malaria is severe we have further to include *Ceylon*.[11] The disease in that island is endemic not only on the coast, but also in the mountainous regions of the interior, even at Njuwera Ellija, situated at a height of 2000 metres [6000 feet]. Another malarious territory is formed by the richly-watered plains and the hilly countries of Lower India, where the fevers occur endemically in their severest forms in *Chittagong*[12] and *Araccan*,[13] in the upper and lower basins of the Irawaddy (*Burma* and *Pegu*),[14] at the mouth of the Salwen (*Moulmein* and *Martaban*),[15] in *Malacca*[16] and the neighbouring islands, in the lower valley of the Menam, especially at Bangkok, and in the plains of *Siam* and *Cochin China*,[17] abundantly watered by the Cambodia and Saigon rivers. Endemic foci of malaria are met with also in wide distribution throughout the *Indian Archipelago*,[18] especially in the *Nicobars*,[19] on the western and southern coasts of *Sumatra*[20] (particularly Singkel, one of

[1] Kinnis, Arnott, Report in 'Bombay Med. Transact.,' n. s., vii, p. 252.
[2] Williamson (I). [3] Gibson (II). [4] Inglis (I), Waller.
[5] Kearney. [6] Broughton. [7] Crespigny.
[8] Young (I). [9] Gibson (II). [10] Young (II).
[11] Marshal (I), Cameron. [12] Macdougall, Beatson. [13] Burnard, Stevenson.
[14] Walsh, Dawson (I), Murchison, Stewart. [15] Day (IV).
[16] Ward and Grant.
[17] Richaud, Fournier, Olivier, p. 55, Thil, p. 18, Veillard, Bernard, Morani, Danguy, Breton (II), Jacquet.
[18] Heymann, Popp, v. Leent (I). [19] Fontana, p. 57, Steen-Bille, i, 244,
[20] v. Leent (IV).

the unhealthiest places in the tropics, Padang and the Bay of Pulo), in *Banka*[1] and the small islands near it, in *Java*,[2] especially its northern and western coasts (Batavia, Onrust, Buitenzorg,[9] Samarang,[4] Sourabaya, Madura, and Banjuwangi[5]), in *Bali*, in *Borneo*, especially on the east and south[6] coasts, and to a lesser extent on the west,[7] on the east coast of *Celebes* and in the *Moluccas*, particularly Amboina, where a focus of intense malaria has developed in more recent times;[8] the *Andaman Islands* belong also to the malarious spots of this part of the world.[9] On the other hand, we have to note certain points which enjoy a comparative immunity from malarial fever; such as the north coast of *Celebes* (Macassar and Kema), *Ternate*,[10] the flat banks of the Palembang in *Sumatra*, the archipelago of *Riouw-Lingga*,[11] and *Manilla*.[12]

Australasia and the Pacific.—An extremely interesting contrast to this wide prevalence of endemic malarial disease in India and the Indian Archipelago is afforded by the state of matters in the *Australian Continent* and throughout Polynesia. Truly endemic seats of malaria are met with there on the coast of *New Guinea*, according to the accounts of Dutch physicians, but nowhere else. Cases of malarial fever are said to have been often observed also in some of the small island-groups, such as the *New Hebrides*,[13] and the *Tonga Group*.[14] The continent of *Australia*,[15] again, so far as it has been hitherto settled by Europeans, particularly its southern and eastern coasts, with *Tasmania*,[16] enjoys an almost absolute immunity from those diseases; and that applies also to *New Zealand*,[17] according to the unanimous reports of observers, as well as to *New Caledonia*,[18] and the *Fiji*,[19] *Samoa*,[20] *Wallis*,[21] *Society*,[22] *Gambier*,[23]

[1] Hollander. [2] Engelbronner, Pecqueur, p. 33. [3] Swaving.
[4] In 'Arch. de méd. nav.,' 1868, Dec., p. 406.
[5] Ib., 1868, Feb., p. 85. [6] v. Leent (II). [7] Reeder.
[8] Epp, Lecocq, v. Hattem (I, II). [9] Hodder.
[10] v. Ewyk, v. Hattem (III). [11] v. Overbeck. [12] Taulier.
[13] Bennet (I), De Rochas, p. 15. [14] Wilkes (III), p. 32.
[15] Richardson (II), Bourse (II). [16] Dempster (I), Scott, Hall.
[17] Johnson, Thomson (I), Bourse.
[18] Vinson (II), p. 16, De Rochas, p. 15, Bourgarel, Charlopin, p. 16.
[19] Messer. [20] Turner, Wilkes. [21] Raynaud. [22] Dutroulau, p. 56.
[23] Account in the 'Arch. de méd. nav., 1876, July, p. 12.

and *Hawaiian Islands*.[1] Brunet, who lived for five years in various parts of Oceania, states that he did not observe a single case of malarial fever during that period. The exemption from malarial diseases of Australia and Oceania affords some important indications for arriving at the pathogenesis, and I shall return to them in the further course of these inquiries.

We find the statements here made to be confirmed, for several of the above-named places, by the statistics of sickness among the British troops in the Australian Colonies and in New Zealand. Among 6786 European troops in Australia from 1859 to 1866 there were 31 cases of malarial fever; and among 43.578 troops in New Zealand over the same period, there were 181 cases. But those cases occurred only among bodies of troops who had been brought direct from India, and had doubtless acquired the fever there. For the years 1867–69, 25 cases were observed among 4491 European troops in Australia and New Zealand, and a single case among 3302 black troops.

China and Japan.—The last malarial region on the continent of Asia, and one of the most intense, is met with in the tropical and subtropical parts of *China*.[2] Not only are there foci of malaria on the coast, among which Macao, Hong Kong,[3] Canton and neighbourhood, Tai Wan (Formosa), Chee-Foo,[4] Shanghai,[5] Chusan, and Tien Tsin may be mentioned as especially unhealthy, but they exist also in the interior, where, as Wilson states, the disease occurs along the course of the rivers as extensively and in as severe forms as on the coast, and where, as he adds, it exerts an influence more pernicious than in the malarial regions of India. More particular accounts of this endemic prevalence of severe malaria in the interior of China have not come to my knowledge, except for Pekin, and more especially for its vicinity.[6] Pernicious malarial fevers are said to be met with besides on the coast of *Corea* and in the southern parts of Manchooria, particularly Fungkiang.[7] On the other hand, in the Russian settlement on the island of Vladivostock (43·6° N.), it is stated by Maurin (following Dr. Aloproff, the Russian

[1] Chapin, Gulick, Le Roy de Méricourt (I).

[2] Wilson (I), pp. 20, 49, 122, 130, Le Roy (II), Gordon (II), Rochefort, Dudgeon, Durand-Fardel.

[3] Macpherson (III), Dill, Smart. [4] Rochefort.

[5] Duburquoy, Henderson. [6] Morache, Rochefort. [7] Watson.

physician of the station) that no case of malarial fever had occurred within a period of five years.

From 1859 to 1866, among 20,858 British troops (Europeans) stationed at Chinese ports, there occurred 11,620 cases of malarial sickness, or 55·7 per cent.; in the years following, from 1867 to 1875, the proportion of malarial sickness was 2203 among 7584 European troops, or nearly 29 per cent., and among black troops 1687 out of 4366 men, or 38 per cent. of the effective force.

As to the endemic occurrence of malarial fevers in *Japan*, there are merely occasional notices from Nagasaki,[1] Yokohama,[2] and Jeddo,[3] and more particularly from the islands of Sikokf and Kiushiu, situated in the south; but it would appear that the disease occurs only to a moderate extent and in its milder forms.

Among 3067 British troops stationed at Japanese ports from 1864 to 1867, 536 cases of malarial fever were observed, but almost all of them among troops that had come from China; in the years from 1868 to 1871 the number of admissions for malarial fever fell to 22 among 2476 men.

Respecting other countries on the continent of Asia situated within temperate or cold latitudes, there are only a few references to the occurrence of malarial diseases at certain places in *Siberia*, such as the mines of Smeinogorsk (51·9° N., district of Kolüwen),[4] Barnaul,[5] and the Barabinsky Steppe, which abounds in marshes and salt lakes.[6]

Europe.—From the point last mentioned, which joins on directly to the Kirghiz Steppe, we pass to European soil by way of *Russia in Europe*, and therewith enter upon a wide region of malaria, which extends from the Steppes of Asia to the Steppe lands of the Caspian, follows the course of the Volga through Astrakhan,[7] and includes the central Caucasian plain and the countries bordering the Black Sea on the north, *Taurida*,[8] the *Crimea*, with the notorious valley of Inkermann,[9] *Cherson*,[10] and *Bessarabia*,[11] the basins of the Dnieper and Dniester, as far as *Ekaterinoslav*,[12] the *Ukraine*,[13] and *Volhynia*,[14] as well as *Moldavia*, *Wallachia*, *Bulgaria*, and

[1] Friedel (I), p. 24. [2] Duburquoy, p. 17. [3] Wernich, Maget.
[4] Rex. [5] Gebler. [6] Woskesensky.
[7] Herrmann, Meyersohn. [8] Milhausen. [9] Heinrich.
[10] Andrejewsky. [11] Tcharnkowsky, Heine. [12] Sachs.
[13] Walter (I), Bulgakof, Gutteeit. [14] Tcharnkowsky.

Hungary, forming the Danubian basin. The malarial fevers prevalent throughout this great territory are well known under the various names of Dacian, Taurian, Crimean, Wallachian, and Hungarian fever, and they have been long of evil repute; even at the present day they may be met with throughout many of the above-named regions in their old pernicious form. A second and less important malarial region of *Russia* extends from Volhynia across the marshy level of Western Russia;[1] and there are still other and smaller foci of endemic malaria in Tula,[2] in Jaroslav[3] (subject to periodical inundations of the Volga), in Orenburg,[4] Samara,[5] Kasan,[6] at a number of places in the Baltic Provinces of Russia, and in the Government of Novgorod where lakes and marshes abound.[7] The last mentioned may be reckoned the most northerly point in Russia to which endemic malarial disease extends. Coming to *Poland*, I find more particular accounts of endemic malaria only for the Government of Augustowo,[8] which has very numerous lakes.

In *Galicia*[9] also, we find endemic foci of malaria in only a few of the smaller districts, especially in the hilly northern part of the country, covered with marshes and ponds, the department of Cracow, and the circles of Wadowice, Zolkiewo, and Zloczow (Brody); while the southern part of the country, rising in terraces towards the Carpathians, is little affected by the disease. A like exemption from malarial disease is enjoyed by the southern slopes of the Carpathians. It is when we descend into the plain that we come upon one of the largest and most notorious malarial regions of Europe, following the course of the Danube and its tributaries from the plain of Lower Austria, extending on both sides of the river over a great part of Hungary, through the low country of Slavonia and Croatia, as well as through the Banat, Syrmia, and the Danubian Principalities, and joining on directly, as we have seen, to the great malarial region of Southern Russia. This area begins with the great *Danubian plain of Lower Austria*, extending from Krems along the river banks to Kornneuburg, and thence, widening out considerably and taking the name

[1] Gorski, p. 12. [2] Koch. [3] Scholvin.
[4] Maydell. [5] Ucke, p. 150. [6] Erdmann, pp. 150, 250, Blosfeld.
[7] Bardowsky. [8] Gorski, p. 11. [9] Schultes, Seidel, Warschauer, Weber.

of *Marchfeld*, to the Hungarian frontier.[1] There it unites with the lesser plain of Hungary, a flat country covered with lakes and extensive marshes, and equally subject to endemic malaria; on the south it is bounded by the Bakonyan Forest, itself the seat of an endemic malaria, while on the nearer side it is continued in one direction through the counties of Tolna and Barany,[2] as far as the marshy shores of Lake Platen, and in another direction it merges with the *great plain of Hungary*. That plain is bounded on the east by the metal mountains of Transylvania, on the north by the slopes of the Carpathians, and on the west by the Danube; it is traversed by the Theiss and its tributaries and covered by extensive swamps, and has long been noted for the prevalence of pernicious malarial sickness, the Dacian or Pannonian fever of history.[3]

Malaria is widely endemic under the same circumstances in the marshy regions of *Croatia*, in the Danubian plain and in the damp valleys of *Servia* and *Montenegro*,[4] in the *Banat*, and in *Syrmia* (valley of the Save), where the annual sickness in several of the districts most infested by the disease appears to reach as high as 30 per cent. of the population, while many low-lying places are scarcely habitable;[5] further, in the river valleys of *Wallachia* (especially the Dobrudscha) and of *Bulgaria*, and in the marshy plains and valleys of *Moldavia*.[6] In many of the countries here mentioned the malarial diseases make themselves felt far up into the mountainous districts.

In the *Balkan Peninsula* we meet with foci of endemic malaria in many parts of *Roumelia*,[7] on the *shores of the Black Sea* and of the *Sea of Marmora*,[8] in *Albania*,[9] and upwards along the coasts of *Dalmatia* and *Istria*; from the latter we have more particular accounts of the disease in Budua, the marshy plain of Pastrovich, Cattaro, Ragusa, Pola, Citta Nuova, marshy spots on the Draga canal and on the Arsa,

[1] Eberstaller, Moller. [2] Lach, Lautz, Entz, Scholz. [3] Wutzer, i, p. 319.
[4] Boulogne. [5] Müller (J), Wenmaring, Lambl, Weinberger.
[6] Tcharnkowsky, Dobronrawow, Seidlitz, Witt, p. 45, Barasch, Dumbreck, Schmalz, Blaustein, Landesberg, Champouillon, Leconte, Obédénare, Dehio, Unterberger.
[7] Witt, p. 45, Rigler, i, p. 376. [8] Beyran, Thirk. [9] Rigler, l. c.

and in Pirano and Capo d'Istria.[1] As regards *Greece*,[2] we are assured of the endemic occurrence of malarial disease at many points in Bœotia and (Attica, Levadia, Locris, the swampy shores of Lake Topolias, Thebes, the country round Athens) Zeituni, Naupantos, and Vonitza (Acarnania and Aetolia), at Chalcis in Euboea, in the Peloponese at Corinth and neighbourhood, Vostiza (the ancient Aegion), Tripolitza, Mistra, Navarino, Modon, and many other places on the coast. In *Crete*[3] endemic malaria is very common, as it is also in several of the *Ionian Islands*, particularly Cephalonia, St. Maura and Corfu;[4] while *Malta* enjoys a complete immunity from malaria except at a few isolated centres near the marsh of Puales, and the frequently inundated La Marsa.[5]

Among 83,835 British troops stationed at Malta during the years 1859 to 1875, there were 428 admissions for malarial fever; in several of those years there were small epidemics of the fever.

In the *Apennine Peninsula*[6] there are especially two great regions that form the seats of endemic malarial disease—the *Plain of the Po* and its tributaries, and the *West Coast* from Pisa down to and including most of Calabria. The first of these begins in the low-lying parts of *Piedmont*,[7] in the provinces of Vercelli, Novara, Lomellina, and Biella, and extends thence through the *Plains of Lombardy and Venetia*,[8] following the course of the Po, and of the Ticino, Adda, Oglio, Mella, Adige, and other of its tributaries, through the Milanese territory, the neighbourhood of Pavia, the country of Siccomario situated between these districts and the west of the province of Lomellina, through the flat

[1] Verson, Erdl, Packley, Trogher, p. 59, Baxa, Wiener, Jilek.

[2] See Faure, p. 47 ff, Mott, Olympios, Pallis, Landerer.

[3] Rosenfeld.

[4] Hennen, p. 219, Ferrara, Black (II), Burnett, Tully, Davy, ii, cap. 10.

[5] Tully, p. 469, Hennen, l. c., Horner (I), Early, Davy, l. c.

[6] For a general statement see Corradi, p. 69 ff. Pareto remarks ('Sulle bonificazioni, risaje ed irrigazioni del regno d'Italia,' Milano, 1865, p. 220), that there are 1,088,961 hectares of marsh land in Italy, of which 65,000 belong to the (quondam) States of the Church, 260,000 to the provinces of Venetia and Milan, and a very considerable extent to Neapolitan territory.

[7] Maffoni, Fossati.

[8] Guislain *passim*, Valentin, pp. 117, 141, Savio (I), Ferrario, ii, p. 299, Hildenbrand, Menis, i, p. 130, Tassani, Lippich, Rossi, Agostini, Donati, Pozzani.

parts of the provinces of Como, Bergamo and Brescia, Cremona and district, Mantua, Verona, Padua, and Ferrara, and thence across the extremely marshy plains that lie between the mouths of the Po and the Marecchia (at Rimini), among which the Valli di Comacchio are specially notorious for fevers. The second great malarial region of Italy, that of the west coast, begins with the marshes on both sides of the Arno, near its mouth, extending from Pietra Santa downwards by Pisa to Leghorn.[1] To the south of Volterra and Siena the district merges in the *Tuscan Maremma*, which extends to Civita Vecchia; this plain, bounded on the east by the slopes of the Apennine, contains hardly any marsh, and is for the most part dry and barren, but it is notorious for its endemic malaria, which is at its worst in the province of Grossetto.[2] At Civita Vecchia, itself a hotbed of malarial fever,[3] the Maremma merges in the *Campagna di Roma*,[4] which, together with the city of Rome,[5] forms one of the chief seats of the disease.

Next come the *Pontine Marshes*,[6] extending along the foot of the hills from Velletri to Terracina, and lastly the malarial region on the *Neapolitian West Coast*, which includes the *Terra di Lavoro* (with the marshes of evil repute in the neighbourhood of Capua), and the provinces of *Napoli*,[7] *Principato citeriore*,[8] and *Calabria*.[9] Smaller malarial spots are met with also on the Adriatic coast, particularly in the neighbourhood of Chieti (Abruzzo citeriore),[10] on the coast of Bari,[11] and at several points on the Gulf of Tarento. Endemic malaria is widely diffused in *Sicily*,[12] not only on the coast or in the plains, but also in the elevated districts. The same applies to *Corsica*,[13] especially the east coast, and to *Sardinia*.[14]

[1] Valentin, pp. 82, 95, Palloni.

[2] Koreff, Marmocchi, Salvagnoli-Marchetti, Danesi, Savio (II). [3] Jacquot (II).

[4] Valentin, p. 54, Griffith, Folchi, Guislain, 37 ff, Fourcault, Armand (II), Colin (I, II), p. 26.

[5] Baglivi, p. 51, Valentin, p. 54, Puccinotti, Bailly, p. 127, Folchi, Guislain, l. c., Fourcault, Bérard, Jacquot (III), Carrière, Armand, Gason, Balley (June), p. 345, Barndel, Colin, i, ii, p. 55, Aitken.

[6] Lancisi, Guislain, p. 43, Fourcault, Sotis, Palestra.

[7] Dorotea, De Renzi, p. 60.

[8] Ely. [9] Hugi, Mamoni. [10] Vicentini. [11] Vitantonio.

[12] Boyle (II), Irvine, p. 3, Zimmermann, p. 13.

[13] Vanucci, Gouraud, p. 29, Abeille, Bennet (III). [14] Moris.

In Ajaccio there are, among 500 French soldiers, 100 annual admissions for malarial fever, not reckoning recurrences. In recruiting for the army in Corsica, out of 1000, representing less than half the number of all who were liable, 774·73 were found to be unfit for service, and these had been rendered unfit for service mostly by severe malarial illness (Costa).

For the *Iberian Peninsula* I am able to adduce only a few general facts about endemic malaria, owing to the absence of more particular accounts.[1] The fevers occur in their severest forms, and to the greatest extent, in the southern and western coast regions; in the low country of Andalusia, on the marshy banks of rivers, especially the Guadiana and Guadalquiver, as well as on the flooded plains of the Tagus, Sado, Mondego, and other coast rivers of Portugal, on the level coast of Granada and Murcia, and the plains of Algara and Alemtejo. Next in frequency, and in less severe forms, it occurs on the dry tablelands of Castile and Estremadura (as at Madrid and Merida), among the mountains separating those two provinces, on the bare coast of Galicia and Asturia, as well as in Barcelona, Valencia, and many other towns on the east coast. Gibraltar, built on rock, enjoys an almost absolute immunity from malarial diseases; out of a total strength of 82,228 men who had been in the British garrison during the period from 1859 to 1875, 657 cases of malarial fever were observed, and 145 of these occurred in a single year. It remains to mention the *Balearic Islands*,[2] especially Majorca, as a region severely infested by malaria.

On *French* soil endemic malaria, apart from its prevalence at numerous isolated spots on the damp banks of rivers or in deeply-cleft, water-logged mountain valleys, is limited more particularly to the western and southern parts of the country. The western region of malaria begins in the lower basin of the Loire, and extends to the mouth of the Adour, or to the foot of the Pyrenees. Upwards from the Loire mouth the endemic habitat extends on both sides of the river through Nantes,[3] Angers, the arrondisement of La Flêche,[4]

[1] Proudfoot (I), Guthrie, Thiéry, i, pp. 238, 270, ii, pp. 12, 17, 159, Wilkomm, Cuynat (I), Martinez y Montez, pp. 494, 497, Pacheco, Leitao, Wallace, Brandt.
[2] Weyler. [3] Bonamy, Anizon. [4] Morisseau.

and Duretal[1] as far as Tours,[2] thence, through the *Sologne*[3] country to the swampy plain of *Brenne*,[4] in the basin of the Indre, not less celebrated than the Sologne for the deplorable ill health of its inhabitants. The endemic region at the mouth of the Loire connects with that of the *Vendée*,[5] the marshy soil of *Charente inférieure*[6] (including the long-known malarial centres (*Marais salantes*) of La Rochelle, Rochefort, Bronage, St. Agnant, and Marennes), the *Gironde*,[7] and lastly the plain of *Landes*,[8] where the malarial region extends westwards to Nerac, and southwards to Dax and Bayonne, or to the slopes of the Pyrenees and the banks of the Adour.

The second great malarial region of France stretches along the *coast of Languedoc and Provence*, with their numerous lakes and marshes. The disease begins to show itself prominently in Narbonne,[9] Beziers, Cette, Montpellier,[10] and Nismes, but the endemic fevers reach their highest point, whether as regards extent or severity, in the Rhone delta—on *Camargue*,[11] and in the level country on both sides of the river mouths (including the malarious spots of Aigues-Mortes, Martigues, Marignane, and others), and in the Palus de Monteux in the department of Vaucluse. Up the Rhone, also, as far as the confluence of the Ardèche, malarial diseases are widely spread ;[12] and we meet with still another and larger centre of them at the confluence of the Saône, in the marshy plains well known by their names of *Dombes* and *Bresse*,[13] which stretch away from Lyons[14] between the Saône and the

[1] l'Humeau.
[2] Heyfelder, Charcellay-Lagarde.
[3] Tessier, Becquerel, Bardel, Boullet, Lafont.
[4] Rigodin, Gizot, Bertrand (H), Gaudon, Hellaine, Godinat.
[5] Bonté, Moureau, Bouquet, Fleury.
[6] Lucadou, pp. 5, 137, Retz, Godelier, Crouigneau, Cornay, Gaillard.
[7] Gintrac, Burquet, Le Gendre.
[8] Dufau, Mondineau, Faye, Sorbets, Lavielle.
[9] Cafford.
[10] Barthez.
[11] Bourely, Boillau-Castelnau, Soumeire.
[12] Madier.
[13] Nepple, Vouarin, Rollet, Beaugrand, Roux, Magnin.
[14] Marmy et Quesnois, pp. 120, 185, 554. Account in the ' Comptes rend. de la Soc. de Méd. de Lyon,' 1840, p. 113.

Ain; and from these we may follow endemic malaria into the country lying between the Saône and the slopes of the Jura as far as Auxonne. Of smaller malarial centres on French soil, there deserve to be mentioned the richly watered plain in Auvergne extending between the mountain ranges of Cantal and Forez, and well known by the name of *Limagne*,[1] and the *marsh country around Lake Indre*,[2] in the department of Meurthe.

In *Switzerland*, where there were formerly many small spots of endemic malaria in damp river valleys (of the Rhine, Linth, Reuss, &c.), and on the shores of lakes (especially the lakes of Zürich and Lucerne) the disease occurs now endemically at only two points, in the southern part of the Canton Ticino and in the Canton Vallais along the Rhone, especially from Sion to its inflow into the Lake of Geneva.[3]

In the south-western parts of *Germany* we meet with small and narrowly circumscribed foci of the disease on the marshy banks of rivers or lakes and in damp mountain valleys (as in the side valleys of the Neckar in the Black Forest);[4] but, besides these, there are larger malarial regions *on the banks of the Rhine* (in Lower Alsace),[5] in the Palatinate,[6] and the Rheingau,[7] and in the *low grounds of the Danube* and its side valleys in Würtemberg[8] and Bavaria.[9] In *Austria* it is again along the Danube that we find the chief seats of endemic malarial disease, although there are also smaller malarious spots in the river valleys of Upper Austria,[10] Salzburg,[11] Styria,[12] and Carinthia;[13] where the river widens out at Krems, we come upon that great region of disease which extends, as we have seen, along its shores to

[1] Monfalcon, Ninet et Aguilhon.

[2] Assalon, Lefèvre.

[3] Lombard.

[4] Rösch (I), Leube, Ludwig.

[5] Renaudin, Cuynat (II), Hahn, Stoeber et Tourdes, p. 403, Wasserfahr.

[6] Pauli, p. 163, account in the 'Bayr ärztl. Intelligenzbl.,' 1854, p. 426.

[7] v. Franque, Lanz, Blümlein, in the 'Vierteljahrschrift für gerichtl. Med.,' 1878, xxviii, p. 100 ff.

[8] Majer (I), Volz.

[9] Schröder.

[10] Streinz (I).

[11] Ozlberger (I, II).

[12] Waser, Onderka, Weiglein, Maeher.

[13] Hussa.

the Black Sea. In *Central Germany*, the disease as an endemic is confined to a few small districts. In the plain of *North Germany*, on the other hand, it is much more widely spread, being found in the basins of the Vistula, Oder, Elbe, Weser, and Rhine. The prevalence of malarial diseases is not inconsiderable even in the *delta of the Vistula*, in *Lower Silesia*,[1] and at a few places in the *Mark* of Brandenburg and in *Mecklenbury*;[2] but it reaches its maximum, both of extent and severity, in the western coast districts of *Holstein* and *Schleswig*[3] (especially Dittmarsh), on the coast belt west of the Elbe, the moor lands of *Hanover*[4] and *Oldenburg*,[5] the damp and in part water-logged low grounds of *Westphalia*,[6] and in the plains of *Rhenish Prussia*[7] bordering the Rhine and its tributaries.

This malarious region of the plain of Germany is continued without break across the *Netherlands*[8] frontier, where the disease is mostly found in the provinces of Grönland, Friesland, and Zeeland with their brackish marshes (the so-called "polders"), and in the coast belt of the provinces of North and South Holland; it is endemic also in the provinces of Drenthe and Overyssel, and in fact no province of Holland is altogether free from it. This malarial area on the west coast of the country merges in the endemic fever region of the low-lying parts of *Belgium*, particularly West Flanders with its numerous marshes, and also East Flanders and Antwerp;[9] whereas the elevated and dry provinces of Brabant,[10] Namur,[11] Liege and the like, are little affected by malaria, and the mountain districts proper are quite free from it.

[1] Lorinser, Klose, Graetzer.

[2] Helm, Brückner, Accounts in 'Beitr. Mecklenb. Aerzte,' pt. i, p. 1, pt. ii, pp. 1, 19.

[3] Hannaeus, Lüders, Friedlieb, Dohrn (I), Dose.

[4] Lauts, Gittermann, Toël, Miquel. [5] Goldschmidt, Wenzel.

[6] Drüfel, Nicolai, 'Sanitätsbericht von Westfalen f. d. Jahr 1838,' p. 86.

[7] Steiffensand, p. 145 ff.

[8] Sebastian, Thijssen (I), Rombach (I), Nieuwenhuys, v. Geuns, Report in 'Algemeene Statistiek van Nederland,' Leyden, 1871, ii, p. 159 ff, Beduin.

[9] Gouzée (I), Meynne, 270 ff, Janssens, Keuwer, Pattyn, Woets, Vraneken, Waldack, Luyx, Puytermans, Thijs, Titeca.

[10] Severon.

[11] Sovet.

The *British Islands* enjoy a very notable immunity from endemic malarial disease, particularly *Ireland*[1] and *Scotland*[2] (which is now at least quite free from it), and the *northern counties of England*[3] and *Wales*. The only localities in which the disease is endemic to any considerable extent occur on the *east coast*,[4] including the *East Riding of Yorkshire*, the counties around the Wash noted for their Fens (Lincoln, Huntingdon, Cambridge, and Norfolk), where, however, the fever has lately decreased to a great extent,[5] and the counties of *Essex* and *Kent*.[6] In the rest of England we meet with only isolated and narrowly circumscribed spots of malaria, mostly associated with damp or water-logged river banks, as on the banks of the Thames in Surrey,[7] and in the South Marsh of Somersetshire.[8]

In the islands of the kingdom of *Denmark*, where malarial fever was formerly reckoned among the prevalent diseases, it now occurs as an endemic sickness only on the islands of Laaland and Falster.[9] It is still meet with in *Norway* as an endemic on the Hvalöer islands (at the entrance of the Christiania Fjord), and in the neighbourhood of Frederikstad.[10] In *Sweden* the foci of malaria appear to have increased considerably in extent and in number of recent years. The disease is found as an endemic at three principal points:[11] in the central depression of the country around the shores of the great lakes, especially Lake Mälar and Lake Wener, the most northern limit of its diffusion there being the Hedemora district (Fahlu-Län) in 60° 20 N.;[12] on the east coast of Torhamn near Hudiksvall (62° N.), being most developed in Kalmar-Län; and at the mouths of several coast streams such as the Angermanna-Elf, the Dal-Elf, and the Götha-Elf. Malarial fever is not endemic in *Finland*,

[1] Wylde. [2] Christison. [3] Proudfoot (II).
[4] Royston, Watson (I). [5] Grantham.
[6] 'Report of the Med. Officer of the Privy Council,' 1859, p. 35.
[7] Hicks. [The Essex shore of the Thames (Barking, Grays), is still a seat of malaria; Surrey and Kent have ceased to be so in any especial sense].
[8] Peebles, Symonds.
[9] Otto, Bremer, 'Sundhetscoll. Aarsberetning for 1876.'
[10] Kjerulf, Broch.
[11] Bergmann, p. 139.
[12] Hallin, Hjelt, Estlander.

nor has it been observed in the *Faröe Islands*[1] or in *Iceland*[2] apart from imported cases.

In the *Western Hemisphere* endemic malarial fever of the severest type has its principal seats in the West Indies, on the Mexican Gulf coast, and in Brazil; but considerable regions of fever, though of a less intense kind, are met with in the northern parts of the Pacific coast of South America, and in the southern, central, and prairie States of the Union.

West Indies.—Among the *West India Islands*[3] those chiefly affected by malarial sickness are Cuba,[4] Jamaica,[5] San Domingo,[6] Guadaloupe,[7] Dominica,[8] Martinique,[9] Sta. Lucia,[10] Grenada,[11] Tobago and Trinidad[12]; while others, such as Antigua, St. Vincent,[13] and Barbadoes[14] enjoy a relative immunity, and the last of these is even in high repute as a sanatorium for patients with malarial sickness. In the *Bahamas*[15] malarial fever is comparatively rare; in the *Bermuda* group it is almost unknown.[16]

In the Bahamas, from 1867 to 1873, there were 305 cases of malarial fever among 1676 black troops stationed there (or 19 per cent.); whereas in Bermuda, from 1859 to 1875, 48 cases of malarial fever were observed among 24,941 European troops (or 0·2 per cent.), and most of these did not originate there, but in the West Indies.

South America.—One of the worst centres of malaria is on the *East Coast of South America*, including the very un-

[1] Manicus, Panum.

[2] Schleisner, p. 2, Finson.

[3] For general information see Chisholm (I), p. 32 ff.

[4] Sullivan.

[5] Sloane, p. 14, Hunter (II), p. 57, Jackson (III), Arnold.

[6] Desportes (I), pp. 52, 93, &c.

[7] Dutroulau, p. 30, Pellarin (II), Manceaux, Carpentin, Batty-Berquin, Raimond, Napias.

[8] Imray.

[9] Savarésy, pp. 33, 51, Dutroulau, p. 30, Saint Vel, Pellarin (I), Rufz, Manceaux.

[10] Wright (II), Evans (II), Levacher, p. 111.

[11] Chisholm (II).

[12] McCabe.

[13] Hunter (III).

[14] Schomburgk, Jackson (IV).

[15] Cleveland.

[16] Account in the 'Sanitary Report on the Colony of Bermuda for 1872,' Hamilton, 1872.

healthy ports of Carthagena, Maracaybo, and Puerto Cabello, and the ill-reputed country of *Guiana*,[1] where the fever is a terrible scourge to the inhabitants, not only on the coast, but also, and even still more, on the inland plains and in elevated situations.

Of 33,486 patients admitted during ten years into the hospital at Cayenne, 16,451, or 50 per cent., were suffering from malarial diseases, and in one year (1855) 13,423 kilogrammes of quinine were used at St. Marie de la Comté, among a force of about 650 men effective (Chevalier). In British Guiana, the number of admissions for malarial fever among the garrison (as averaged for the period from 1859 to 1863) was 77 per cent. of the total strength; according to Blair's account, quinine is used there to the amount of 20 grains annually per head of the population.

A region of less intense, but very widely spread malaria, covers almost the whole of the *north of Brazil*[2] as far down as Rio de Janeiro; and here also the disease is equally prevalent in coast localities and elsewhere—on the flat and often inundated banks of the Amazon, Rio Madeira, Maranhão, Paranahyba, San Francisco, Parana, Rio Doce and their tributaries, on the island of Santa Catarina, and in the marshy districts (some of them elevated) of the provinces of Piauhy, Para, Mato Grosso, Goyaz and Minos Geraes. There are also widely diffused endemic foci of malaria in the *prairie lands* (pampas) of *Paraguay*[3] and *Bolivia*,[4] particularly in the provinces of Tucumana, Salta and Santa Cruz. The circumstances are decidedly more favorable in the southern provinces of Brazil, San Paulo and Rio Grande do Sul, and that applies still more to *Uruguay* and the eastern provinces of the *Argentine Republic*, which, according to the unanimous verdict of observers, enjoy an almost absolute immunity from malaria.[5] On the Pacific coast of South America,

[1] Bajon (II), p. 20, Campet, p. 81, Segond, Laure (I), p. 7, Dutroulau, pp. 18, 250, Lauzach, Chevalier, Maurel (for Cayenne), Schöller, Hille, Popp (for Surinam), Rodchsied, p. 215, Blair (for Brit. Guiana).

[2] Sigaud, pp. 157, 216, Lallemant, Gardner, Rendu, p. 67, St. Hilaire (II), p. 50, Jobim, Plagge, account in 'Gaz. med. da Bahia,' 1868, May, p. 15, Bourel-Roncière, Rey (II).

[3] Mantegazza (I), p. 286, (II) pp. 80, 224, Masterman.

[4] Bach.

[5] Dupont, p. 13, Brunel, p. 36, Mantegazza (I), p. 100, Féris (II),

Chili, which was formerly quite exempt from malaria,[1] has been visited since 1851 by pernicious epidemics, and at a few points in that country the disease has assumed an endemic character;[2] but the proper region of severe endemic malaria does not begin before *Peru*, where the disease occurs very abundantly and in severe forms equally on the coast and among the deep eastern valleys and spurs of the Sierras.[3] The endemic sickness extends thence along the coast to *Ecuador*,[4] and probably also to *New Granada*; in the eastern parts of Ecuador, especially in the districts within the upper basin of the Maroñon, malarial diseases are said to be rare.[5]

Central America.—In the countries of *Central America*,[6] the malarial diseases have their chief seat on the Atlantic (Gulf) coast from Chagres up to Cape Garcias a Dios, and on the interior plain up to a height of 600 metres. The Pacific coast is less severely visited, although there also endemic foci of malaria are met with, particularly in Corinto (port of Nicaragua),[7] on the coast of San Salvador and in the valleys of the Lempa and St. Miguel,[8] as well as on the coast of Guatemala.[9] In *Mexico*, also, it is on the Atlantic coast that malaria predominates, as in several ports of Yucatan (Belize in British Honduras,[10] Sisal and Carmen), on the coast of Tabasco,[11] in Alvarado, Sacrificios, San Juan d'Ulloa, Vera Cruz,[12] Tampico and Matamoros (Tamaulipas), along the banks of the Rio Grande del Norte,[13] and elsewhere.[14] It is met with also as an endemic, although on the whole less frequently, at many points in the Sierra Templada as high as 1200 to 1500 metres (as in Orizaba[15] and Oaxaca[16]); it is only on the table-land proper (Anahuac) that it vanishes entirely,

[1] Lafargue, v. Bibra (I), Boyd. [2] Piderit, Le Roy (III).

[3] Hamilton (I), Tschudi, Le Roy (II), pp. 180, 188, Fournier.

[4] Le Roy (II), p. 280. [5] Galt.

[6] For the Isthmus of Panama: Lidell, Buel, Le Roy (II), p. 285, Wagner (II), p. 20, and Schwalbe; for Costa Rica: Le Roy (II), p. 373, v. Frantzius, and Schwalbe; for Nicaragua: Bernhard, Le Roy, p. 376, Watson (II); for the Mosquito Coast: Young (III).

[7] Gibbs. [8] Guzman. [9] Durant, Bernouilli.

[10] Hamilton (II). Of 4045 British troops (black) 1008, or 25 per cent., sickened of malarial fevers during the years 1859 to 1873.

[11] Morel, Heller, Müller (II), Jourdanet, p. 150.

[12] Naphegyi, Heinemann (II). Vaillant and Corre do not admit the endemic prevalence of malarial fevers in Vera Cruz. [13] Pouillé, Pirion.

[14] Debout, Pommier, p. 37. [15] Thomas (I). [16] Uslar, Heiremann (I).

so that at the elevation of the city of Mexico malarial fever is observed only as an epidemic now and then.[1]

On the *Pacific coast of Mexico* the circumstances as regards endemic malaria are the same as for the adjoining coast of Central America; here also the endemic fevers are confined to a few points, among which may be mentioned Acapulco, Tepic, and the strip of coast from San Blas to Mazatlan.[2]

United States.—Beyond the Rio del Norte, this great malarial region extends over the whole *Gulf coast of the United States* as far as the Cape of Florida, spreading far into the interior of the continent along the Colorado, Brazos, and Mississippi, and their tributaries.[3] In *Texas*[4] the malarial region stretches from the coast and the swampy banks of the Rio del Norte, Nueces, Colorado, and the smaller coast streams up into the highlands, where foci of severe sickness are met with as high as the upper basin of the Colorado (Fort Duncan in Eagle Pass), and at Fort McKavit at a height of 600 metres. The disease appears to be still more widely diffused in *New Mexico*, being met with at elevations of over 2000 metres in Fort Bayard, Fort Union, and other places, and forming a terrible scourge to such of the native population of the country as inhabit the damp valleys. The limit of its endemic prevalence here is Santa Fé (2300 metres), where malarial fever is no longer met with.[5] From the western part of the *Louisiana* coast,[6] between the Sabine and Mississippi, the malarial region extends across the zone of bluffs in that State, over a great part of *Arkansas*, particularly along the banks of the Mississippi and Arkansas rivers, and over the marshy plains in the north-east of the country, stretching away towards Missouri, and still farther along the Arkansas river over the eastern part of the *Indian territory*, including the malarious spots of Fort Gibson[7] (noted as the "charnel-house of the army)," and Fort Sill.

[1] Newton, Jourdanet, p. 387, Liberman.

[2] Celle, p. 26, Caddy, Girard, pp. 13, 18.

[3] See particularly Bartlett, p. 345, Drake, and the 'Statistical Reports on the Sickness and Mortality in the Army of the United States' (in synopsis of the literature, 'Reports' II).

[4] Meyer, Bracht, Neufville, Moses (I), Rösch (II).

[5] See 'Reports' (II), 1860, pp. 213, 218, 223. [6] Collins, Gibbs.

[7] Wright (III), Coolidge, Sinks (on the endemic malaria of Kansas).

Malarial disease is endemic at only a few scattered points in the great prairie land of this territory, which rises towards the Rocky Mountains from south-east to north-west, and has a dry sandy soil, not often saturated by any heavy falls of rain. In like manner, the eastern part of Louisiana, beyond the Mississippi, forms part of a region little subject to malaria; this region includes the coasts and the hilly zone of the State of *Mississippi*,[1] and in particular the Pine Woods, so much reputed for their healthiness, a range of moderately high sand-dunes, covered with fir woods, which begin at Lake Pontchartrain and run along the coast at no great distance inland, as far as the Bay of Pensacola, intersected by the Pearl River and by the Pascagoula, Perdido, and Alabama. These Pine Woods are much resorted to by the inhabitants of New Orleans and Mobile when malarial fevers and yellow fever are prevalent. On the belt of hills in Mississippi State, as far as Vicksburg, malarial endemics are met with at isolated points only, as for instance, on Grand Gulf, which is notorious for its fever; but from Vicksburg there spreads out along the valley of the Yazoo a great swampy plain rising towards Memphis, noted for the endemic prevalence of severe malaria ("Yazoo swamp fever"). The eastern hill region of Mississippi is well situated as regards healthiness, and that is the case also with the greater part of the sparsely populated State of *Alabama*[2], in which malaria is endemic chiefly on the coast, especially around the swamps on the Bay of Mobile, on the banks of the Alabama and Black Warrior (counties of Wilcox, Dallas, Lowndes, Montgomery, and Tuscaloosa), and on the marshy plain of Huntsville, lying to the south of Tennessee and reaching to the borders of that State. In the Peninsula of *Florida*,[3] the sickness is widely diffused on the Gulf Coast with its jungles and swamps, particularly in the counties of Escambia (including Pensacola) and Gadsden, at Tampa Bay, Fort Meade, and other places; the same is true also for part of the Atlantic coast,[4] but in a lesser degree; for the health there, especially

[1] Wharton, Ferrar, Montgomery.

[2] Lucas, Heustis (I), Boling, Levander, Bates, Wooten, Capshew.

[3] Porter (I), Little, Gaillard (I), 'Reports' (II), 1856, pp. 309, 336.

[4] 'Reports' (II), 1860, pp. 148, 151, 164.

in St. Augustine, appears to have improved materially in recent years. But the chief seat of malaria in this State is formed by the plains of the interior, partly swamp, and also by the plateau of no great elevation which forms the water-shed for the Bay of Tampa, and runs up the peninsula to Georgia. In *Georgia* the disease prevails widely and in severe forms,[1] not only in the numerous creeks of the coast, but also in the interior, the neglect of agriculture in quite recent times having greatly conduced, as it seems, to an increase both in the amount and intensity of the endemic fever.

In the Central States of the Union, malaria is endemic to an extent that is still considerable, though materially less than in the Southern States; and chiefly on the coasts of *South Carolina*,[2] *North Carolina*,[3] *Virginia*,[4] and *Maryland*,[5] and on the damp river banks of the interior. Improvement of the soil has helped not a little to narrow the range of the endemic in these States; but it is rather remarkable that malarial fevers have increased within the last twenty years in localities formerly little touched by them, especially in the mountainous districts of Virginia.[6] In the inland Central States of *Tennessee*[7] and *Kentucky*,[8] malarial fever occurs to a moderate extent along the banks of the Mississippi and Ohio, attaining its greatest prevalence on the prairies of the latter State known as "the barrens." On the prairie States proper,[9] *Ohio, Indiana, Illinois, Missouri, Iowa, Minnesota, Wisconsin*, and *Michigan*, malarial fevers are likewise widely prevalent, diminishing in frequency, however, towards the north west. Thus, in the northern parts of Iowa the fever is no longer so common as in lower

[1] Le Conte, Daniell (I), Pendelton, Posey, 'Report of the Board of Health of the State of Georgia,' Atlanta, 1876.

[2] Simons, Collins, Gaillard (II), Porter (II).

[3] Williamson (II), Norcom (I), Norcom (II), McKee, Manson, Dickson, Winborne.

[4] Somervail, Thomson (II), Perkins. It is stated by Bland that Virginia west of the Alleghanies is quite free from the disease, except at a few endemic malarious spots on the banks of the Ohio.

[5] Beatty, Stille, Reyburn.

[6] Simons, McKee, Perkins.

[7] Ramsay, Becton, Higgason, Buchanan, Hogg, Harper, Cunningham, Tuck Grant, Bailey.

[8] Yandell.

[9] Bradford, Parry, Carrol, Hewins, Cook Farnsworth.

latitudes under the same circumstances of tillage; and that is the case also in Wisconsin and Minnesota, where the rate of malarial sickness among the troops (in Fort Snelling, 42·52° N., and in Fort Ripley, 46·10° N., both on the Mississippi) amounts to about 15 per cent. only; still more is it the case in the territories of Dakota and Montana, where the rate falls respectively to 5 per cent. and 6 per cent. The largest foci of disease in those regions are met with on the shores of the great lakes; and here, again, the geographical situation proves to have the most decided influence on the occurrence of malarial sickness. Thus, the shores of Lake Superior, and in part, also, those of Lake Michigan and Lake Huron, are entirely free from fever; it is not endemic, for example, at Winnebago, Wisc., in latitude 44° N., notwithstanding marshes and a damp river bank, and it is comparatively rare in the swampy settlement of Fort Brady.[1] It is in the southern parts of the State of Michigan[2] that we come upon the true domain of malaria, and we then follow it along both shores of Lake St. Clair to the junction with Lake Huron, and along the southern shores of Lake Erie and Lake Ontario as far as the St. Lawrence. Detailed accounts from that region speak of pernicious malarial fevers at Fort Gratiot, Detroit, Plymouth, and other places on the United States side, and at Amherstbury, Fort Malden, Sandwich,[3] &c., on the Canadian side. Even on the northern side the range of sickness on Lake Ontario extends from Hamilton to Kingston, and still farther up the ridge which runs along the shore from Burlington to the mouth of the Trent, attaining in some places a height of more than 600 feet.[4]

These lake-shore endemics of fever extend also to the north-western parts of the State of *New York*, although there are many localities in the counties of Onondega, Tompkins, Seneca, Ontario, Oneida, and others, formerly much subject to fever, that have now become tolerably free from it owing to improvements in the soil.[5] It is mostly along the banks

[1] Reports (II). [2] Sutphen, Beech. [3] Doucet.

[4] Mendenhall, Drake, i, p. 334, 'Reports' (II), 1859, p. 30, Sprague, Stratton, Kerr.

[5] Fribre, Brown (II), Hart, Smith (III).

of the Hudson, and on a narrow strip of the coast, that the sickness is endemic in this State; but within the last twenty or thirty years a remarkable increase of fever has been noted in the counties situated among the mountains.[1] The same thing has been observed also in *Pennsylvania;* as the disease has retired from places that used to be its head-quarters, such as the country bordering the Schuylkill, the Susquehanna, and the Delaware, it has come to be more prominent in the mountainous districts of the State.[2] It must remain an open question how far improvements in the soil have contributed to this decrease of malaria in its old foci; at the same time it is undoubted that it has been observed to disappear from localities where no changes in the ground have taken place. The latter circumstance obtains, in part at least, for *New Jersey* also, where there has been a remarkable decrease of malarial fevers within recent years in many localities that used to be visited by it severely.[3]

In the *New England States* malarial fever is endemic at only a few points;[4] in the State of Maine it is no longer endemic.[5]

Neither is it endemic throughout the greater part of British North America. For *Canada*, as well as for the whole inland basin of the continent, Kingston (44·8° N.) is the northern limit of endemic malaria; as an epidemic, one meets it at higher latitudes, on the banks of the St. Lawrence and its tributaries, on Lake St. Peter, very rarely at Montreal or Quebec, or places on the coast such as Halifax[6] (N. S.) and Miquelon (N. F.) in the latitude of 46·30° N. The cases in *Nova Scotia* and *New Brunswick* are imported ones. In *Greenland* malarial fever is quite unknown.[7]

In the western regions of North America the limit of

[1] Smith (III), Trask, 'Reports' (II), 1840, pp. 130, 133, 1856, p. 14.

[2] Rush, p. 97, Parrish, Accounts in the 'Transact. of the Pennsylv. State Med. Soc.,' 1862, 1865, 1867, 1868.

[3] Accounts in the 'Transact. of the New Jersey State Med. Soc.,' 1861, 1862, 1868.

[4] 'Reports' (II), 1840, pp. 110, 114, 117, 121, 125, 1856, p. 9, 1860, p. 10, Threndwell (for Boston). See p. 232.

[5] Wotherspoon, 'Reports' (II), 1840, pp. 142, 146, 1856, pp. 27, 30.

[6] Boyle (III).

[7] Lange, p. 32.

malaria reaches to somewhat higher latitudes. It is prevalent there chiefly on the slopes and in the valleys of the Rocky Mountains[1]—whence the name of "mountain fever"—in the territories of *Wyoming, Utah,*[2] and *Colorado,* and it is especially disastrous to the Indian tribes.[3] Only imported cases occur at Fort Vancouver (*Washington Territory*)[4] in latitude 45·40° N., and on the *Oregon* coast,[5] as well as in *Alaska*[6] (formerly belonging to Russia). Not until *California*[7] do we reach a more considerable malarial region on the west coast; it extends up the valleys of the Sacramento and San Joaquin; and in the inland southern part of the State (Arizona) malarial fevers appear to be widely prevalent. But the sub-tropical coast of Southern California from Monterey to San Diego enjoys a noteworthy immunity from the sickness,[8] being in that respect similarly situated with the Pacific coast of Mexico and Central America.

§ 61. Epidemics and Pandemics—Old Seats and New—New Types.

The area of distribution of malarial disease here sketched in general outlines will have to be considerably extended if we take into account not merely the endemic occurrence of the sickness, as we have hitherto done, but also those regions in which the disease appears only now and then as an epidemic. These *epidemics of malaria,* which extend not unfrequently over large tracts of country, and sometimes even over whole divisions of the globe forming true *pandemics,* correspond always in time with a considerable increase in the amount of

[1] Smart (II).

[2] Ewing, Bartholow, Waggoner, Brewer.

[3] Wilkes (IV), p. 369, Gairdner, Moses.

[4] Maurin.

[5] Glisan.

[6] Blaschke, p. 62; during a five years' residence at New Archangel he saw only three cases of malarial fever.

[7] Stillman, Blake, Hammond, Praslow, p. 44, Gibbons, Logan, Keeney (in 'Reports' (II), 1860, p. 243.)

[8] King, Summers (in 'Reports' (II), 1856, p. 438), Biggs and Graves (in 'Transact. of the Californ. State Med. Soc.' for 1870 and 1871). According to the account of Hoffmann (ib.) malarial sickness is endemic in San Diego itself.

sickness at the endemic malarious foci, whether near or distant; they either die out after lasting a few months, or they continue—and this applies particularly to the great pandemic outbreaks—for several years, with regular fluctuations depending on seasonal influences.

On the very verge of the period to which the history of malarial epidemics can be traced back, we meet with a pandemic of that sort in the years 1557 and 1558, which is said to have overrun all Europe.[1] It is impossible to decide from the scanty and incomplete epidemiological data of the sixteenth and seventeenth centuries how often such epidemic outbreaks of malarial fever may have recurred in times subsequent to that pandemic; it is not until the years 1678-1682 that we again meet with definite facts relating to an epidemic extending over a great part of Europe; and thereafter follow at short intervals reports of the same kind for the years 1718-1722, 1748-1750, 1770-1772, and for a more restricted epidemic in 1779-1783. Although malarial fever during the last ten years of the previous century and the first five years of the present had absolutely disappeared from the arena of national pestilences, and had even diminished considerably in those places where it was endemic, there developed in 1806 a pandemic of malaria which overran a large part of the north and north-east of Europe, lasting till 1812; it coincided with an epidemic of malaria in Southern India in 1809-1811, which extended from the slopes of the Mysore mountains to Cape Comorin, and from the Western Ghâts to the Coromandel coast. During the ten years following, malarial fever was again confined within its habitual limits; but thereafter, a little earlier or a little later in the several regions, there arose one of the most extensive, severe, and persistent of pandemics, beginning in 1823 and dying out in 1827, of which there are numerous medical reports from almost all parts of the world. The next general epidemic prevalence of malarial fever falls in the years 1845-1849, after which comes the great pandemic of 1855-1860. Finally, we have the malarial pestilence of 1866-1872, in which the disease spread not only over a great part of Europe, but visited simultaneously many parts of

[1] Palmarius, 'De morbis contagiosis,' lib. vii, Paris, 1578, p. 322.

India (Presidency of Madras, Lower Bengal, Punjaub, &c.) and of North America, and showed itself for the first time, and that too in a severe form, in the islands of Mauritius and Réunion.

Decrease or disappearance.—To complete this account of the historical and geographical aspects of malarial disease, we must first of all observe that in many parts of Europe and North America it has become of recent years not only less frequent than in the previous century, but also less severe in type. Pernicious malarial fever was prevalent as late as the eighteenth century in many parts of Germany, in the Harz, in Augsburg, Saxony, Silesia, Würtemberg, and other localities where now it occurs only in occasional epidemics, and then always in its mildest forms. At the time of Sydenham and Willis and of Huxham, London and Plymouth were dangerous fever spots, whereas to-day malaria is a rare thing in them; and the same applies to Stourport,[1] Bolton,[2] and other towns in England. In Scotland, where there were still many endemic malarial foci remaining in the eighteenth century,[3] the disease is now extremely rare. It is the same in Ireland, where, as Wylde remarks,[4] no acute infective disease is so rarely met with as malarial fever. It is further noteworthy that the disease has become less common and milder in character in the Netherlands, in many parts of Belgium,[5] and at numerous points in the United States of America, particularly in certain counties of Pennsylvania,[6] New York, New Jersey[7] and Maryland,[8] that used to be much subject to fever; in some of the Southern States also, such as Florida,[9] the disease has assumed a decidedly milder form.

Fluctuations.—Not less striking than this gradual sub-

[1] Watson (III). [2] Black (I).

[3] Christison. Wilson (II) observes that, whereas in the ten years from 1777 to 1787 the annual number of malarial cases in Kelso was one seventh, and sometimes even one fifth of the total sickness, it had fallen in the ten years from 1829 to 1839 to one six-hundredth.

[4] 'Edin. Med. and Surg. Journ.,' lxiii, p. 263.

[5] Meynne, p. 284.

[6] 'Trans. of the State Med. Soc. of Pennsylvania,' 1856-60-62-65-67-68-71-72.

[7] 'Transact. of the State Med. Soc. of New Jersey,' 1861-62-68.

[8] Worth.

[9] Gaillard (I).

sidence and disappearance of the disease are the fluctuations observed, at the several places, in the amount of the sickness, partly connected no doubt with the already-mentioned pandemic outbreaks of malaria, but to some extent independent of these. Another noteworthy circumstance is the development of endemic foci of malaria at places that had been hitherto quite exempt, or only occasionally visited by epidemics.

Thus, to mention only a few of the facts: a widespread outbreak of malarial fever appeared in 1823 at Prague, where the disease had not been known for years; it continued until 1830, when it again became very rare, and it did not receive any considerable fresh accession until 1846.[1] At Stuttgart, where malarial fevers are counted among the diseases most rarely observed, the sickness, after being epidemic in 1826 and having been completely extinguished, broke out still more extensively in 1834,[2] and showed itself in the very same year at other places in Würtemberg occupying elevated and dry situations. At Königsberg (Province of Prussia), where the conditions of the soil are very favorable to malaria, the sickness was scarcely observed at all from 1811 to 1825, but after that an epidemic of it developed which lasted until 1833; from 1833 to 1841 the disease recurred in isolated cases only; from 1841 to 1852 it appeared every year in the spring to a moderate extent, but from 1852 to 1855 it was prevalent to an extent and of a severity that one but rarely sees in so high a latitude.[3] Observations to the same or corresponding effect have been made at Marienwerder,[4] Leipzig,[5] Erlangen[6] and other places in Europe; also at the more intense centres of malaria, as many facts from tropical countries prove.[7]

New Foci.—A phenomenon not less interesting meets us in the fact often observed in more recent times, of new foci of malaria being established, or of its epidemic continuance for several years, and its wide diffusion, in localities which had previously been quite free from it, or at least practically free from it.

One of the islands of the Indian Archipelago, Amboina, had, until the year 1835, enjoyed a remarkable immunity from malarial sickness; but in that year a severe epidemic arose, it is said in consequence of an earthquake that took place at the time, and since then the island has been a permanent seat of pernicious malarial fever, and has conse-

[1] Bischoff (I), p. 31. [2] Cless (II). [3] Hirsch.
[4] Heidenhain. [5] Thomas (II). [6] Küttlinger.

[7] In the Presidency of Madras, the number of deaths from malarial disease from 1868 to 1871 rose from 105,692 to 132,346, 151,027, and 193,398, or almost to double within four years ('Madras Monthly Journ. of Med. Sc.,' 1872, v, p. 298).

quently become one of the most unhealthy places in the East Indies. The East African islands of Mauritius and Réunion experienced the same fate in 1866; they had previously been almost exempt from malarial fever, but in that year a disastrous malarial epidemic developed, and its persistence to the present time makes it probable that endemic foci of the disease have been established.[1] In Chili, where malarial fever was formerly almost unknown, the disease showed itself first in 1851 as an epidemic, and it now appears to have become domiciled at several places in that country.

It is pointed out by Perkins that, in the eastern parts of Virginia, where severe malarial fever used to be very rare or sporadic, it has greatly increased in extent and frequency within the last ten years (the report dating from 1845); and, according to the account of Trask, the same fact has been observed in the county of Winchester, N.Y., where, about the year 1848, malarial foci sprang up in several districts hitherto entirely exempt, while at the same time there was no especial increase of the sickness to be made out in the neighbouring districts in which it was endemic. Within the last ten years similar observations have been made also in Pennsylvania and the New England States. In Connecticut, says Burrows, malarial fever was common at the time of the first English settlement; with the increasing cultivation of the soil, it disappeared almost entirely, being met with only in a few river valleys. In quite recent times, however, it has again become somewhat common, and it gets more and more widely diffused. This reappearance of malarial fever dates from the year 1866; New Haven was the centre, and in the years following down to 1872 the disease spread all round to Fair Haven, East Haven, Bradford, Guilford, North Haven, Hamden, and Meridan. Several observers in Connecticut have pointed out that the disease showed itself and got diffused at several points coincidently with the making of railway cuttings, the excavation of canals, and such like earth-works; but we may take it that these are not the only circumstances in which the essential cause is to be sought, for the reason that malarial fever has subsequently shown itself in localities where that etiological factor is not available, and further by reason of the fact which many practitioners vouch for, that numerous other forms of disease have assumed a character peculiar to and typical of malarial sickness, and have proved amenable to treatment by quinine in a much more marked way than formerly. From this Burrows concludes that a kind of malarial diathesis has developed among the inhabitants, and in that way the general prevalence of the disease is to be accounted for. Also in the north of Europe, there has been noted at several points a striking increase of malarial fever within the past ten or twenty years; particularly in Sweden and Finland, where, according to Hjelt, the

[1] 'Reports of the British Army,' 1867, ix, p. 101, 1868, x, p. 105, 1869, xi, p. 119, 1870, xii, p. 104, Barraut, Borius (II), Small and Power, Account in the 'Lancet,' 1868, Feb. 22nd, p. 264, Mercurin, Barat, Edwards, Stone, Rogers, Welch, Nicolas, Blaxall, Tessier, Lacaze, Bassignot, Monestier, Labonté.]

disease occurred first in epidemics limited to the south-western parts of the country, but has penetrated since the beginning of the present century, farther and farther towards the north and east.

New types.—The occurrence within the last twenty years of a severe form of remittent malarial fever, described under the names of *fièvre bilieuse hématurique, hæmorrhagic malarial fever,* or *febris remittens hæmorrhagica,* is an interesting phenomenon in this connexion. This form of fever has been observed in various malarious localities in the tropical and subtropical regions of the Eastern and Western Hemispheres. There is no doubt that the malady, distinctively marked as it is by hæmaturia due to renal affection and by more or less intense jaundice from severe liver disease, had been observed by the practitioners of former times. We find indications of this in the descriptions which have been given by the French of "fièvre bilieuse grave" and by the English and Americans of "bilious remittent fever;" but the disease has been frequently confounded with yellow fever, and it is only of recent years that we have arrived at a complete knowledge of it, owing, doubtless, to the increased range over which it has shown itself and the general attention that it has attracted.

The first exact data about this disease come from Madagascar and the Comoro Islands, from which we have accounts by Daullé (II), Monestier (I), and Borius (II). Later information on the same subject is furnished from Senegambia by Barthélemy-Benoit, Bourse (I), Chabbert, Serez, Léonard, Berenger-Férand, Verdier, Rey (I), and Defaut; from the Sierra Leone coast by Gore; from Gaboon (Guinea Coast) by Forné and Abelin, and by Dudon and Falkenstein from the Congo Coast. In Réunion and Mauritius, also, according to the statements of Monestier (II) and Labonté, many cases of that form of disease were observed in the severe epidemics of malaria which have occurred there since 1866. For the continent of Asia, there is a single notice of its occurrence in India (Day, 'Indian Annals of Med. Sc.,' 1859, Jan., p. 105), and others from Cochin-China by Disser and Veillard.[1] On the other hand,

[1] V. Leent mentions a "febris remittens biliosa" in Sumatra, which was observed in Europeans only; it may perhaps be counted with the above form of severe malarial sickness.

this form of disease appears to have attained a very wide diffusion in the West Indies (according to Pellarin (I, III, IV), Rufz, Manceaux and Raimond for Martinique and Guadeloupe, Sullivan for Havana, and Gibbs for Nicaragua) and still more in the Southern States of the Union. Almost all the authorities in those regions speak of it as a disease hitherto unknown, or at least very rarely observed. In Texas it showed itself first in 1866 (Ghent, Tate, Starley, Hewson, Johnson (II), Heard), and about the same time on the coast and in the central swamps of Louisiana (Barnes, Délery, Faget); thereafter in the State of Mississippi as far up as Natchez (Sharpe, in the 'Transact. of the Mississippi State Med. Soc.' for 1874), in Arkansas (Duval in the 'Transact. of the Arkansas State Med. Soc.' for 1871), and in Alabama, where it is prevalent to a very considerable extent, according to accounts by Kinnard, Scholl, Osborn, Michel, Riggs, Hendrick, Weatherley, Anderson, Mabry, and Webb; it has lately been reported to occur also in North Carolina by Raleigh and Greene.

The observations do not point as we have said, to a new form of disease, but to an increase in the amount or frequency of sickness; or, in other words, to the prominence assumed by several forms of malarial fever in places where they had previously occurred only as isolated cases, and had for the most part so escaped the attention of the observers. This applies above all to the diffusion which the malady has attained within recent years on United States' soil. There is only one account hitherto pointing to the occurrence of this form of disease in the malarial regions of Europe, viz. one from Sicily; in 1877, Tomaselli published observations that he had made in Catania of severe sickness in patients who had suffered for a long time from malarial fever and had used large quantities of quinine, and he ventured to regard these complications as a result of quinine poisoning. When the communication was made to the Academie de Médecine, it was pointed out by Le Roy de Méricourt that the group of symptoms described by Tomaselli formed a complete picture of "febris remittens hæmorrhagica," and we find that this interpretation of the facts has been adopted by Mancini and Marotte in their most recent communications.

We may here refer to still another form of malarial fever, which was first described by the United States' physicians under the name of *typho-malarial fever*, and which attracted the general attention of the medical world on its appearance among the American troops during the war of Secession. According to the account of Woodward, the disease showed itself first in the Federal Army in the autumn and early winter of 1861. The surgeons, who mostly came from the Northern States and were well acquainted with the phenomena of typhoid fever, were surprised to find in it a form of disease which had hitherto been strange to them. The course of the malady is sketched as follows by Woodward, from his observations in the Army of the Potomac.

In those cases in which the malarial affection predominated, the disease presented itself in the form of a simple intermittent or remittent; not until after seven to ten days did the fever become continued, or the phenomena peculiar to typhoid show themselves,—diarrhœa, abdominal tenderness, meteorism, delirium, dry and brown tongue, and the like. But it happened not unfrequently that the symptoms peculiarly characteristic of typhoid were wanting, such symptoms as the diarrhœa and the rose-coloured spots, while pain in the region of the liver and a slight degree of jaundice were more frequent than in ordinary typhoid. Many of these cases ran a favorable course, especially under large doses of quinine, but deaths were not unfrequent. Post-mortem examination showed, as a rule, only a simple catarrhal affection of the mucous membrane with swelling and pigmentation of the solitary follicles and the Peyer's patches, and sometimes swelling of the villi in the small intestine with pigmentation at their apices. Corresponding changes occurred here and there in the large intestine. Next in order, enlargement of the spleen was often found, and congestion of the liver with or without fatty degeneration. Histological examinations of the lymphatic follicles in these cases brought to light changes such as accumulation of lymphoid cells, and sometimes their impaction in the nearest lymphatic vessels and in the connective tissue, which differed from the changes occurring in typhoid only in degree. Again, in those cases where typhoid infection was predominant, the disease took essentially the form of typhoid, and the post-mortem examination showed in a marked manner the changes proper to that disease. But the disease in question was characterised by the marked periodicity of its course; the periodicity had often the typical intermittent character, becoming most pronounced in the defervescence and at the stage of commencing convalescence. Further, the enlargement of the liver and spleen was characteristic of malaria, and was not found in the same degree of development as in simple typhoid; and finally, deposits of pigment (melanæmia) occurred in various tissues, as in malarial fevers.

Earlier hints of the occurrence of this peculiar form of disease in the United States may be found in the descriptions of its malarial fevers given by Drake, Dickson, and Wood; researches on the same subject have been recently undertaken by Clymer, Flint, Loomis, and others, most of whom share the opinion of Woodward that we have not to deal here with a peculiarly modified form of malarial fever, but with a hybrid affection, a combination of malaria and typhoid.

The same form of disease has been described quite recently by Borelli for Naples, Aitken for Rome, Obédénare for Wallachia, Durand-Fardel for Chinese ports, and Maurel for the French convict settlement on the banks of the Marañon (Cayenne). The description which these authors give of the course of the disease, corresponds in the main with the sketch of it by the United States' surgeons, but in their views of its nature they differ from the latter; Borelli, differing from most of his Neapolitan colleagues, who have expressed themselves in the same sense as Woodward, holds to the view that the disease represents a modified form of typhoid; Aitken, again, declares it to be a peculiar malarial fever, and Obédénare, Durand-Fardel, and Morel would also make it out to be a severe form of malarial sickness. The latter is the opinion expressed by Van der Burcht in his description of a fever observed by him at Gonda, which likewise showed the symptoms of a mild typhoid, but had remissions and intermissions in the latter part of its course, and proved thoroughly amenable to quinine and Fowler's solution. Finally, Colin in his essay on 'Typhoid in the Army' also declares against the hybrid character of this disease; he holds it to be rather an unique malady, the result of a transformation of malarial fever into typhoid. The data available at present as to this "typhoid malarial fever" afford no certain clue to its nature; we may expect some enlightenment upon it in the immediate future, now that the attention of physicians has been directed to it at many points.

An explanation of the fluctuations in the amount of malarial disease at the several parts of the globe will be discussed in connexion with the question how far those diseases reach to in their geographical distribution, and upon what factors their limitation depends.

§ 62. Regions of Intermittents, Remittents, and Malarial Cachexia.

By far the most frequent and most widely distributed *form of malarial disease* is the *intermittent malarial fever*, which is met with at all times and in all places wherever and whenever the disease is endemic or epidemic; in some circumstances it represents, along with remittents of a mild type which very often change in the end into the intermittent form, the malarial process exclusively. This is true, above all, of the disease where it occurs endemically in higher latitudes; and it is for the most part true of the epidemics of malaria in the same regions, and still more of epidemics where the disease is not indigenous, the severer forms (remittent and pernicious fevers) being more likely to occur where the malady is endemic. This (intermittent) kind of malarial sickness accordingly, is met with most in the central and northern regions of Europe, on the rocky north coast of the Iberian Peninsula and the plateau of Spain, in the mountain districts of Upper Italy, in the tropical and subtropical regions of Asia little affected by the disease in general (Aden, Singapore, northern parts of China,[1] Japan, the southern slopes of the Himalaya),[2] on the table land of Abyssinia,[3] on the prairies of the River Plate States (especially those of Paraguay and Entre Rios), and of Australia, in many of the mountain valleys and on the tablelands of Peru, New Mexico, and Texas, over the greater part of California, and, excepting on the shores of the great lakes, in the northern division of the Eastern States of the Union, the region of severe malaria beginning on the other side of New York.

The *severe remittent and pernicious malarial fever* is principally a disease of tropical and sub-tropical countries. Its headquarters are on the West African coast territories, from Senegambia down to the Congo coast, the coast plains and the numerous oases of Algiers,[4] the malarial countries on the

[1] Morache. [2] Curren. [3] Blanc.

[4] Bachon (for the oasis of Laghouat), Audet (for Tuggurt). Sériziat mentions that in an oasis inhabited by about 400 souls, 15 adults and 30 children died of severe malarial fever in *one* year.

East Coast of Africa, as given above, as well as Madagascar,[1] the Comoro Islands, and the lately invaded islands of Mauritius and Réunion, the river basins and swampy levels of Abyssinia[2] and Khartoum, Lower Egypt (particularly the Isthmus of Suez[3]), and Tunis; on Asiatic soil the coasts of Arabia and Beloochistan, the plain of the Euphrates, the malarial districts of India[4] and of Further India, Ceylon, numerous places in the East Indies (especially in Java and on the coast of Borneo), the southern and south-eastern coast belts of China and a large part of its interior, the coast and the marshy plains of Persia[5] and part also of its tableland, Syria, and the coast of Asia Minor and Transcaucasia.

In the Western Hemisphere, the chief regions of remittents are: the coast, the valleys of the Sierra and the wooded region of Peru, Gayaquil, the sub-tropical provinces of the River Plate States (especially Tucuman), some parts of Chili where, as we have seen, a pernicious malarious endemic has lately developed, the marshy coast, plains, and damp river valleys of Brazil and Guiana, the coasts (especially the Atlantic) and damp valleys of Central America, the coast belt of Mexico, the malarious parts of the West Indies, as given above, the Southern and Central States of Union, with part of Pennsylvania, the prairie-lands (especially Illinois,[6] Indiana, and Iowa[7]), the shores of the lakes Huron, Erie, and Ontario within the limits above stated, and many districts on the western slopes of the Rocky Mountains (where the sickness is known as *mountain fever*[8]) including some of the Californian valleys,[9] whereas the coast on that side of the North American continent enjoys an exemption from malarial fever, and especially from the pernicious forms of it.

On European soil the area of endemic prevalence for

[1] Borius (III), Borchgrevink. [2] Blanc. [3] Anelli.

[4] Especially notorious in the hill-districts of the Deccan under the name of "hill fever." See Wright (I), Heyne, Macdonnel, Murray.

[5] See Polak, and the account by Bell of the pernicious malarial fever that prevailed in 1842 from the mouths of the Indus extending in a north-westerly direction up to Teheran.

[6] Hewins, Cook.

[7] Farnsworth.

[8] Waggoner, Bartholow, Smart (II).

[9] Praslow, pp. 47, 49.

the severe remittent and pernicious malarial fevers is a somewhat limited one; they are most widely spread and most frequent in the steppe-lands of Southern Russia, in the Crimea, in the Ionian Islands, in some parts of Greece and of Turkey, in the Danubian Principalities, in the marshy districts and the low grounds of the Danube and the Theiss in Hungary, in the Banat, Istria, and Dalmatia, in the valley of the Po, in the malarious region of the Italian west coast, in Sardinia and Corsica, and on the southern and western coasts of the Iberian Peninsula. In more isolated spots, and on rarer occasions, we meet with this form of the disease as an endemic in the marshy valleys of Styria,[1] in several of the regions of more intense malaria in France (La Vendée, Sologne, Charente, Landes, Bresse, Camargue), in the coast provinces of the Netherlands, in a part of Belgium,[2] in a few marshy districts of Rhenish Prussia, and in the coast belts of Oldenburg, Hanover, and Holstein.

The relation as regards sequence, of these severe malarial fevers to the simple intermittent form, is for the most part such, that at the beginning of the epidemic or the rise of the endemic, intermittent fevers are observed almost exclusively, that in the subsequent progress the cases of severe sickness become more and more numerous, predominating at the height of the endemic, again becoming relatively fewer as the amount of sickness decreases, while only intermittent forms are observed at the close. This ratio of severe forms in the endemic becomes the more pronounced the larger the dimensions assumed by it, or, in other words, the nearer the endemic approaches to an epidemic; and when in such cases there comes to be an epidemic degree of prevalence even in those regions where malarial fever is not indigenous, the severe forms will sometimes show themselves to a considerable extent there also,[3] although it is still in the proper foci of malaria that they predominate.

[1] Onderka.

[2] According to Titeca, it still exists in Antwerp as well as in other places.

[3] At the time of the well-known seacoast epidemics of 1825-27, this relation came out very prominently in the comparatively frequent occurrence of severe malarial fever in Sweden, Denmark, and many of the non-endemic localities of Germany and France; this was also the case at the time of later pandemics (1846-48 and 1855), particularly in Sweden, and also during the epidemic of

As regards the geographical distribution of *the malarial cachexia*, the principle holds good generally that it is most frequently met with in those more intense foci of malaria which give rise to the endemic prevalence of the severer forms of fever. Its development in the individual, however, is by no means dependent on preceding attacks of malarial fevers, but it may arise under the continuous influence of the morbid poison; and it has often been developed, even in individuals who have remained entirely exempt from the disease in its feverish forms. It is the malarial cachexia that contributes so materially to the excessive mortality found in malarious regions.

§ 63. Regions and Circumstances of Quotidian, Tertian, or Quartan Fever.

The form and duration of *the intermittent type of the fever* in the various parts of the globe that the disease frequents, is a question that comes into closest connexion with the inquiry concerning the geographical distribution of the several kinds of malaria. The general answer to this question is that the types with shorter periodicity, approximating to continued fever—the quotidian and duplicated tertian—are proper to tropical and subtropical regions, while in higher latitudes the fevers with more prolonged intervals, viz. the tertian and quartan, are most characteristic.

There is practically but one opinion among authorities as to the prevalence of the quotidian type in the tropics. "In the tropics," says Day (II, p. 74), "the quotidian type assumes the proportion borne by the less fatal tertian in more temperate climes." Laure (II, p. 94) remarks, regarding the tropical colonies of France: "In the provinces occupied by us the paludal fevers are almost always intermittent and of the quotidian type; the tertian type is rare, and still rarer is the quartan." Dutroulau (l. c., p. 154) says: "Among the regular types the quotidian is most frequently recognised; the statistics from all our palustral colonies prove this, and we may regard it as the general type." Frantzius (l. c., p. 326) states: "Costa Rica resembles other tropical countries in having the quotidian intermittent by far the most frequent

1823-27 in the British Isles, in which according to my view, it is with malarial fever, and not, as Murchison assumes, with relapsing fever, that we have to deal.

form of ague; the tertian occurs much more rarely, and it is extremely seldom that one has an opportunity of observing the quartan." Less decidedly pronounced, but still clearly recognisable, this law shows itself also in higher latitudes, particularly in the Southern States of the Union, in the Ionian Islands,[1] in Greece,[2] Turkey,[3] the Crimea,[4] and other parts of Southern Russia (as in Astrakhan, where Meyersohn estimates the quotidian type to be twice as common as the tertian) and even in Istria,[5] the Banat,[6] and other malarious regions of Southern and Central Europe.[7]

A more particular analysis of the facts leads certainly to some conclusions that are in apparent contradiction to this law. But those are just the exceptions which enable us to formulate the principle more exactly; which show, in fact, that *the type of the fever stands in a definite relation to the intensity of the malarial process.* We find, accordingly, that:

1. The tertian type prevails in those regions within the tropics where the milder malarial fevers are indigenous, as, for example, at Aden,[8] the mountainous parts of the East Indies,[9] a few points in the Madras Presidency, and on the Peruvian coast, where the disease occurs in less severe forms than in the valleys of the Sierra or in the forest region.[10]

2. In the malarious regions of the tropics, the natives take the milder forms of the fever, while the foreigners, and particularly those not acclimatised, take the disease in its severer forms; and, in accordance with that fact, the types with longer intervals occur in the former and those with shorter intervals in the latter.[11]

3. The frequency of the quotidian type in endemics or

[1] Hennen, Ferrara.
[2] Faure, p. 49.
[3] Rigler, Dumbreck.
[4] Heinrich.
[5] Verson, Müller (I).
[6] Wenmaring.
[7] In the marshes of Holstein, the types of fever, according to Dose, occur in frequency in the ratio of 20·5 per cent. of quotidian, 51 per cent. of tertian, 26·1 per cent. of quartan, 2·4 per cent. of duplicated quartan, and 0·3 per cent. of pernicious fever.
[8] Howison.
[9] Swaving.
[10] Hamilton (I), Smith (II), Tschudi.
[11] See particularly the accounts of Borius (I) and Chassaniol from Senegambia, the report on the great malarial epidemics of 1809-11 in India, and the special accounts by Nash, Henderson, and Morehead from the same country.

epidemics is in direct proportion to the severity of the disease.

Thus Mondot states that, when the endemic malaria of Senegambia takes a mild form, tertians prevail. The observations published by Shanks on the forms of malarial sickness in an English regiment at Secunderabad from 1837 to 1841 are highly instructive in this respect; in the first year, when the endemic was little developed, all the cases were of the tertian type, while in the three following years, with the endemic at a considerable height of intensity, the quotidian type was observed in five sixths of the admissions. As in the severe epidemics of tropical countries, so also in the recently observed appearance of the disease in Mauritius and Réunion, we always find the shorter type predominating; the same holds good also in part for the severe malarial epidemics in higher latitudes, as, for example, in the sea-board epidemics of 1825-27, and in the epidemic of 1843 at Marienwerder,[1] and of 1859 at Amsterdam.

Finally, we interpret in the same way the fact that

4. In the endemic or epidemic prevalence of malarial fever, the tertian type occurs at the outset, whereas at the height of the endemic or epidemic, or whenever in general it assumes a severe character, the type is quotidian, and as the sickness abates there are again the longer types of the fever, the tertian in tropical and subtropical countries, in higher latitudes often the quartan. We may recognise this behaviour of the disease even in the malarial centres of Charente,[2] of Hungary,[3] the Banat,[4] and of Transcaucasia,[5] although it is more pronounced in lower latitudes such as those of Algiers,[6] Senegambia,[7] and India.[8]

Morehead[9] observes with reference to the forms of the disease in India: "Quotidians will be found to prevail most generally at those seasons of the year when the generation of malaria is believed to be actively going on," and to the same effect are the reports of Day (II), and of Geddes (1)[10] for the epidemic of 1825 at Seringapatam, and for that of 1826 at Caddapah. Concerning the behaviour of the marsh fevers around the Bay of Jahde, Wenzel states:[11] "The facts observed here agree with those observed elsewhere; that is to say, when the morbific agent becomes more intense, as genetic power rises, there is not only a corresponding increase in the average severity of the form of sickness, but also a preponderance of the shorter rhythms and an approximation to the continued type; while, with decreasing intensity in the colder

[1] Heidenhain, p. 523.
[2] Cordier.
[3] Scholz, p. 315.
[4] Weinberger.
[5] Popoff, 1857, pp. 52, 267.
[6] Haspel, Deleau.
[7] Gauthier.
[8] Geddes (II), pp. 94, 134.
[9] P. 22.
[10] Pp. 45, 127.
[11] P. 23.

months, there is a corresponding lengthening of the rhythm to tertian and quartan." I shall have an opportunity of adducing further evidence on this point when I come to consider the question of the comportment of malarial fever in the several seasons of the year.

§ 64. No Race or Nationality immune—Acclimatisation.

Just as the history of malarial disease shows it to have been a malady of all times, so the inquiry into its geography leads us to recognise in it a disease of all *races* and *nationalities*. This predisposition to malarious sickness is developed to the highest degree among all the peoples belonging to the Caucasian stock, not only on European soil, but also among the Arab population of the Barbary States,[1] and in the malarious districts of India, where the Mohammedan and Hindu population[2] suffer in the same degree as the foreigners. This is not less true for the Malay[3] and Mongol[4] stocks and for the native (Indian) population of North and South America.[5] The predisposition is least for the Ethiopian race, which, although it by no means enjoys an absolute immunity from the disease, is still affected by it, *ceteris paribus*, less frequently, less readily, and less severely than other races; and to this many experiences have incontestably testified, not only in Senegambia,[6] the West Coast of Africa,[7] Nubia,[8] and other parts of its native habitat, but also in other malarious

[1] Jacquot (II, p. 265), Espanet, Furnari, and other authorities for Algiers.

[2] Milroy, Huillet, Campbell, Winchester, Leslie, Thil, p. 18, Ollivier, p. 55, Morani, p. 21.

[3] Cameron, p. 71, for Ceylon; Epp, v. Hattem (II), and others for Amboina; Heymann, p. 342, for Singkel; Hodder, for the Andaman Islands; v. Leent (IV), for Sumatra.

[4] Wilson (I), p. 71, Duburquoy, Henderson ('Edin. Med. Journ.,' 1876, No. 405), and others for China.

[5] See the accounts by Praslow, p. 44, and Keeney ('U. S. Army Reports,' 1855-60, p. 243), for California; by Moses (II), p. 38, and De Smet ('Voyage aux Montagnes Rocheuses,' Gand, 1849), for Colombia; by Thomas, Keller, Liberman, and Porter ('Amer. Journ. of Med. Sc.,' 1853, Jan., p. 36), for Mexico; by Rendu, p. 69, for Brazil; and by Tschudi, for Peru.

[6] Thevenot (I), p. 245, Gauthier, p. 116, Chassaniol, Berger, p. 51.

[7] Daniell (II), p. 154, Gordon (I), Monnerot, p. 39.

[8] Pruner, Hartmann.

regions of the tropics whither they have migrated.[1] This relative immunity from malarial fever on the part of the Negro race is an acquired and not a congenital one, as we may learn by the frequent cases of sickness and death from this disease among the children of the negroes in Senegambia. But the same immunity is enjoyed by the natives of *all* malarious regions so far as concerns their own home and such other localities as are affected by malaria less severely than it; so that one might almost formulate a general rule that the predisposition to malarial sickness becomes weaker in proportion as the individual has been continuously exposed, from birth to maturity, to more or less severe malarial influences, without suffering from them to any considerable extent.

Even in the malarious regions of Europe, in the Netherlands,[2] Belgium,[3] Pola,[4] Syrmia and the Banat,[5] Rome[6] and the Ionian Islands,[7] the indigenous population sicken much less frequently and much less severely than persons who have come there from regions free from malaria. The same circumstance is reported from the Caucasus,[8] the malarious centres of the Southern States of the Union,[9] Algiers,[10] Aden[11] and other subtropical parts of the globe; but the fact comes out most clearly in the great malarious regions of India,[12] Ceylon,[13] Further India,[14] the West Indies,[15] Central America[16] and other countries in tropical latitudes. The following is

[1] Moulin, p. 21, and McCabe for the West Indies; Frantzius, p. 328, for Central America; Vaillant for Mexico; Cameron for Ceylon; Heymann for the East Indies.

[2] Blane, 'Observ. respecting Intermittent Fevers,' select dissert., London, 1822, p. 90.

[3] Meynne, p. 273. [4] Erdl. [5] Wenmaring.

[6] Colin (I), Aitken ('Brit. Med. Journ.,' 1873, March).

[7] Hennen, 'British Army Reports,' 1839, p. 34 *a*.

[8] Kaputschinsky.

[9] Le Conte for Savannah, Williamson (II) for North Carolina.

[10] Furnari, Maillot, p. 265.

[11] Steinhauer.

[12] Annesley, McGregor, Stewart ('Transact. of the Calcutta Med. Soc.,' viii, p. 149), Macdougall, Twining (II), p. 207.

[13] Marshall.

[14] Jacquet.

[15] Hunter, Evans.

[16] Liddell, Buel, Horner (II).

a table compiled by Waring[1] of the malarial sickness during ten years among the troops in the Madras Presidency:

	Total strength.	Admissions for intermittent.	Admissions for remittent.	Percentage of the troops.	
				Intermittent.	Remittent.
European troops .	103,431	13,264	4336	12·8	4·2
Native troops .	568,403	95,354	8046	16·8	1·4

The native troops accordingly suffered from simple malarial fever to a greater extent even than the European, but the number of cases of remittent fever observed was three times less among the former than among the latter. In Ceylon there died of malarial fevers per 1000 of the population:

Negroes	1·1
Natives of India	4·5
Malays	6·7
Natives of Ceylon . . .	7·0
Europeans (English) . . .	24·6

Also in the severe epidemic malaria of the Mauritius, according to the account of Barat, the coloured races (Kaffirs and Malagasys) have suffered less than others. In Costa Rica, says Frantzius (p. 328), the severe forms of malarial sickness occur most frequently among the blond and blue-eyed northeners (Europeans) and the residents in the cooler regions of the highlands, while the settlers in the malarious districts and the acclimatised population are affected either not at all or only with slight agues, negroes and mulattoes showing the greatest resistance to the malarial poison.

It is in this sense, then, and notwithstanding all the objections which have been urged on the ground of observations wrongly interpreted, that we may speak of an *acclimatisation* against malarial disease, assuming that the individuals have been exposed to malarious influences for a long time, and have escaped unscathed altogether, or been only slightly affected. But, on the other hand, it must be admitted that nothing so much increases the predisposition to the sickness

[1] P. 460.

in general, and to the severe forms of it in particular, as repeated infection. This is the sense in which there is some reason in the contention of Dumontier,[1] Morel, Lecoq (p. 527), Monnerot (p. 19), Morani (p. 21), and the others, who deny the possibility of acclimatisation against malarial fever, on the ground of observations made for bodies of troops that had been stationed many years in malarious places in the tropics. The view that I have put forward receives a material support from the most recent experiences of the occurrence of severe forms of hæmorrhagic remittent fever in the tropics, according to which, in the nearly unanimous opinion of observers,[2] the sufferers have been almost solely Europeans,[3] or, at all events, have never been the new comers, but only such as had suffered from malarial sickness many times before.

Finally, there is a series of observations to indicate that the immunity gained by acclimatisation will prove insufficient in proportion as the severity of the epidemic increases, that it will be lost again if those who have been acclimatised stay too long in localities free from malaria, that it pertains only to the place in which it was acquired, and that those who remove to other regions of severe malaria will no longer enjoy protection from the sickness.

In evidence of the first mentioned fact, the insufficiency of acclimatisation in severe epidemics, there are many data from India, such as those of Langstaff respecting the great malarial epidemic of 1829 at Delhi and other places in the North-West Provinces. On the second point—loss of immunity in consequence of too long a stay in regions free from malaria,—I adduce the interesting observations of Prichett and McWilliam made in the unfortunate Niger Expedition of 1841-42 : out of 158 negroes who accompanied the expedition eleven sickened of malarial fever, and those eleven were persons who had lived for a long time (in England) at a distance from their native country. Pritchett adds that he had observed the same fact often before in the West Indies,

[1] 'Considérations sur l'acclimatement des Européens dans les pays chauds,' Paris, 1866, p. 28.

[2] Daullé, Bourse (I), p. 17, Serez, p. 38, Abelin, Manceaux, Sharpe, Osborn, Sullivan, Pellarin, Veillard, Falkenstein, Rey (I), Defant.

[3] Scholl has seen only one case of febris hæmorrhagica in a negro, and he had already gone through many attacks of malarial sickness. Of 121 cases of this form of malaria observed by Foncervines in Mayotte, twenty-seven belonged to the coloured race and the rest to the Caucasian.

and Gairdner[1] quotes from Captain Trotter's report on the same expedition, the statement that "the constitution of the negro, whether of African or American birth, requires an habitual residence in Africa to be exempt from the fever of the country." Lastly, for the insufficiency of acclimatisation where there has been a change in the malarious place of residence, there are various interesting pieces of evidence from different malarious countries in the tropics; thus, according to Grierson, Sepoys from India proper sickened on the marshy soil of Arracan in almost the same numbers and to the same degree as European troops; in Burmah, according to Day, two thirds of the Sepoys suffer from the severest malarial fevers; and Beatson[2] observes that Indian troops coming from Hindostan to Chittagong run a great risk from malaria. Natives of St. Louis (Senegambia) who migrate to Bakel, are subject to malaria there almost as much as Europeans; the same fact is stated by Michel (I) with reference to the Senegalese who have migrated to the Gold Coast, and by other observers for negroes from Guinea who come to the Island of San Thiayo—one of the Cape Verd Archipelago, and known as "Mortifera" from its deadly climate—and there incur the same risk as other foreigners.

§ 65. Influence of Climate and Season.

Among the factors which determine the occurrence and diffusion of the malarial diseases, climatic and telluric conditions hold the first place.

The dependence of malaria-production on *climatic influences*, of which the geographical distribution of the disease over the globe has already given us indications, is brought out in the most definite way by the prevalence of malarial fever (1) at certain seasons and (2) under certain meteorological conditions, particularly under the influence of heat and atmospheric moisture.

In those localities where malarial diseases are endemically prevalent, they occur at *all seasons of the year;* but we everywhere meet with maximal and minimal periods of sickness, and it is with the maximal periods that those epidemics are associated which spread beyond the malarious districts to localities where the disease is not indigenous. A comparison of these seasonal periods of maximum and minimum for the several malarial regions, shows many differences among

[1] 'British Army Report' for 1865, p. 335.

[2] Pp. 60, 62.

them, while there is a certain uniformity within particular latitudes. Thus, we find that:

1. In regions with moderately developed malaria, there are two maxima, one in spring and one in autumn, a considerable decrease of the disease in the months between them, viz. in summer, and a minimum in winter.

This kind of incidence of the sickness is met with in many localities belonging to higher latitudes,—in Sweden, Denmark, the north of Russia, Poland, a great part of Germany, and the north of France, and in high-lying or mountainous regions of tropical or subtropical latitudes corresponding to the above, such as the tableland of Mexico.[1]

A closer examination of the circumstances in these cases will convince us that the autumnal maximum occurs oftenest wherever the malaria is most intense, and that the great epidemic or pandemic outbreaks of the disease usually reach their height, therefore, in late summer or autumn.[2] The details summarised in the three following tables furnish striking examples of these ratios in the amount of sickness.

Table of Malaria throughout the Year in Temperate Regions.[3]

	Sweden.[1]	District[2] of Jahde.	Leipzig.[3]	Vienna.[4]	Klagenfurt.[5]	Dittmarschen.[6]
January . .	3149	51	68	257	81	198
February . .	3432	52	118	231	58	212
March . .	5428	55	341	422	64	490
April . . .	8138	61	794	779	83	748
May . . .	8567	61	1292	1110	136	790
June . . .	4889	56	1147	865	115	490
July . . .	2867	56	679	658	111	326
August . .	2749	166	446	698	102	1182
September . .	3731	211	290	846	87	1318
October . .	3779	192	100	702	113	596
November . .	3399	123	63	512	113	334
December . .	2881	69	46	312	78	212

2. In regions with strongly developed malaria, there is a maximum beginning in summer, which reaches its height at

[1] Newton.

[2] This incidence of the disease comes out very clearly in the great malaria epidemics of 1807-11, 1824-27, and 1846-49.

[3] For footnotes to this and the following table see bottom of p. 250.

the end of the summer or the beginning of autumn, lasting not rarely into the winter, and which so far exceeds the spring maximum that the latter not unfrequently disappears altogether, so that there is only one minimum, winter and spring, and one maximum, summer and autumn.

This is the curve of the disease that one meets with mostly in warm or subtropical regions, such as southern and western France, Italy, Sicily, Sardinia, and Corsica, the Iberian Peninsula, Southern Russia, Hungary, the Banat, Dalmatia, Algiers, Tunis, Egypt, Syria, Persia, Transcaucasia, and the Central and Southern States of the Union. In all these countries, the more extensive epidemic outbreaks happen in the summer and autumn months, chiefly from July to October (sometimes as early as June), and they may then last through part of the winter, into December and even later. From numerous observations confirming these statements, I have drawn up the following table giving a series of statistical data for a number of localities.

Table of the Incidence of Malarial Fever throughout the Year in Warm Countries.

	Corbeil.[7]	Szent-Miklos.[8]	Rome.		Pola.[11]	Algiers.[12]	United States of America.[13]					
			In general.[9]	Malignant fevers.[10]			Central.	Southern.	Florida.	Texas.	New Mexico.	California.
January . .	117	234	201	25	2·4							
February . .	133	161	96	5	1·9	35	2709	2902	1637	2864	601	604
March . . .	245	204	81	7	2·0							
April . . .	405	538	80	8	2·4							
May	554	714	99	13	2·8	202	4028	3976	2842	3807	648	686
June	427	512	117	20	2·9							
July	502	566	443	107	8·7							
August . . .	891	963	518	230	14·2	796	5748	7283	2638	5506	1559	1316
September . .	782	795	782	154	12·6							
October . . .	407	561	951	109	11·3							
November . .	216	398	496	42	5·8	455	5247	4658	2381	5653	1435	864
December . .	12·8	274	311	2·8	3·1							

3. Finally, in the most intensely malarious spots of the tropics, the prevalence of the disease is generally associated in a most marked manner with the rainy season; the fever usually makes its appearance with the commencement of the

rains, and lasts through the whole of that period; if the rainfall be not excessive, it reaches its maximum usually when the rains cease, and continues with decreasing extent and virulence, until the setting in of the cool season. But inasmuch as the rainy season in the several regions of the tropics has somewhat different times for setting in and a varying duration according to the geographical position and the configuration of the country, there are also very considerable differences in respect to the time when the sickness is prevalent in the several tropical malarious countries.[1]

Further, the period of fever is protracted much longer here than in the localities of which we have already spoken, not unfrequently, indeed, far into the so-called cold season; and this holds particularly for those regions where the differences in temperature are the least marked between the several seasonal periods, and applies therefore more to the tropical regions of the Western Hemisphere, especially the West Indies and Guiana, than to those of the Eastern. The following table makes clear the conditions of sickness which we are now considering, for a number of points within the tropics.

[1] See McClelland, p. 120, v. Frantzius, p. 319.

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[1] From the reports of the Swedish Board of Health ('Sundhets-Colleg. Berättelse' for the years 1820-73.

[2] According to Wenzel for cases of sickness among harbour labourers in the years 1860-69.

[3] According to Thomas (II), p. 233, from twenty-three years' observations in public institutions for the sick, and in practice among the poor.

[4] Hussa, from ten years' admissions into the General Hospital.

[5] The same, from twenty-five years' observations.

[6] Dose, from observation of 6896 cases during the years 1842-63.

[7] Petit, from observation for twenty years.

[8] Lach for three years (1854-56).

[9] Balley, l. c., May, p. 401, from the admissions at the Military Hospital of S. André, 1858-60.

[10] Baccelli for two years' observations in hospitals at Rome.

[11] Jilek, p. 26. The numbers give the proportion of sickness in the total force of troops for the years 1863-67.

[12] Villette, from several years' observations among the French troops in Metidjah.

[13] From the 'U. S. Army Reports' for the years 1839-59.

Table of the Incidence of Malaria throughout the Year in Tropical Countries.

	Bombay.[1]	Cochin.[2]	Hyderabad.[3]	Mewar.[4]	Madras.[5]	Deccan.[6]	Senegambia.[7]	Guiana.[8]	West Indies.[9]
January . .	6·5	210	955	3·0	4·8	6·34			
February .	4·3	201	821	2·6	2·7	5·53	416	740	2048
March . .	4·9	227	956	2·6	4·1	6·30			
April . . .	5·1	183	994	1·8	4·9	6·68			
May . . .	8·9	222	896	1·1	8·6	5·99	234	774	1303
June . . .	11·9	213	758	1·6	8·1	5·15			
July . . .	10·2	278	1256	2·8	7·5	8·45			
August . .	9·5	295	1719	4·0	14·8	11·65	746	803	2236
September .	7·6	262	1861	6·7	17·0	12·56			
October . .	13·5	305	1668	12·8	12·9	11·12			
November .	10·7	344	1561	8·7	8·8	10·42	936	866	2492
December .	6·8	308	1464	6·0	5·8	9·81			

§ 66. Influence of Heat.

The question here raised leads us to inquire into the influence on the production of malarial disease exerted by those factors that are characteristic of the climate—by the *conditions of heat and moisture*. That inquiry rests directly on our knowledge of the geographical distribution of malaria, and the facts given in the course of the foregoing sketch find a definite expression in the law that the disease shows a progressive decrease both in extent and intensity from the equator to the poles, and that there is a certain limit beyond which it does not occur either endemically or epidemically,

[1] Reports by Stowell, p. 6, and Waring, p. 464, on the admissions for fever into the General Hospital of Bombay, 1838-43, and 1846-56; the figures give the ratio per month in each 100 cases in the year.

[2] Day (V), p. 240, from ten years' observations in the Civil Dispensary.

[3] Day (I), p. 56, from five years' observations among the troops.

[4] Ewart, p. 464; the figures show the proportion of sickness in every 100 soldiers.

[5] Geddes (II), p. 87, from five years' observations among the troops, as in (1).

[6] Day (II), p. 57, also reckoned as in (1).

[7] Dutroulau, p. 9, from two years' observations in the hospital at St. Louis.

[8] The same, p. 18, from the hospital in Cayenne during two years.

[9] The same, pp. 30, 31, from two years' observations in the hospitals in Martinique and Guadeloupe.

or, if it do occur, then only in cases imported. In the Southern Hemisphere, where this limit is found within comparatively low latitudes, the limitation is undoubtedly more dependent upon telluric than upon atmospheric influences. But the circumstances in the Northern Hemisphere must be judged otherwise; here the limit corresponds to a line which starts from 55° N. on the western side of North America, sinks to 45° on its eastern side, rises to 63° or 64° on the western side of the old world (Sweden and Finland) and runs across Northern Asia in about the latitude of 55°. In the following table I have put together two groups of observations, the first of which contains a series of places just within the limit where malarial fever is endemic or epidemic, while the second embraces points that lie beyond the limit and are free from the disease (except as imported) notwithstanding the presence of some factors, notably in the soil, which are favorable to its production.

	Locality.	Latitude.	Mean temp. of the year.	Mean summer temperature.
I.	Quebec (Canada)	46°5	3°0 C.	17°5 C.
	Kingston (Canada)	44°8	6°7	19°8
	Angermanland (Sweden)[1]	63°	3°5	16°7
	St. Petersburg	59°56	3°8	16°0
	Barnaul (Siberia)[2]	53°19	−0°5	17°6
II.	New Archangel (Alaska)	57°3	7°4	13°5
	Fort Ripley (Minn.)	46°10	4°0	15°3
	Fort Kent (Maine)	47°15	2°7	15°2
	St. John's, N.F.	47°32	3°5	13°4
	Julianehaab (S. Greenland)	60°0	−0°4	6°0
	Iceland	64°0	4°1	13°1
	Faröe Islands	62°0	...	10°0
	Haparanda	65°5	−0°25	16°1

From this we may conclude that the summer isobar of 15°—16° C. (58°—60° Fahr.) marks the limit[3] of the occur-

[1] From meteorological observations in Hernösand.

[2] This is the farthest point in Siberia from which there are any accounts known to me of malarial fever occurring.

[3] Wenzel (p. 20), from observations made in 1858-69 among the harbour labourers in the district of Jahde, arrives at the confirmatory result that a temperature of 12° R. (15° C.) is the limit for the development of malaria there in the summer quarter.

rence of malarial fever, and that those regions where the mean summer temperature does not reach that height, are exempt from the disease. A rather high temperature, therefore—and the opinions of all observers agree in this—forms an essential condition for the development of malaria; and one would be justified in ascribing to this factor, within limits at least, the manner of diffusion of the disease over the globe, attributing to the higher temperatures the predominance of the disease in tropical and sub-tropical regions, and to the relatively lower degrees of heat the less frequent, milder, and less persistent forms of temperate latitudes. A confirmation of this is found not only in the circumstance that the extent and intensity of the disease in malarious foci at the several seasons of the year are in direct proportion to the height of the respective temperatures, but also in the fact that the great epidemics or pandemics have been immediately preceded by hot years or have coincided with them.

"The severity of these diseases" (intermittent and remittent fever), says Annesley (p. 39), writing of India, "is generally in proportion to the warmth of the climate or season in which they occur." To the same effect is Morani's opinion from Cochin China: "The observation is made every day, that it is almost always at the moment of greatest heat that the fever declares itself;" and the same opinion is expressed by Serez (p. 38), by Gauthier for Senegambia, by Von Frantzius (p. 519) for Costa Rica, by Caddy for Mexico, by Levander for Alabama, and by Armand (p. 131), Corne, and others for Algiers. Similar observations have been published from temperate latitudes, as, for example, from Rome by Balley, who says (p. 428): "To this period of greatest heat (July and August) there corresponds the greatest pathogenetic elevation in the whole year; the march, so to speak, of the temperature and of the pathogeny is the same;" and this is confirmed by Barudel (p. 118) almost in the same words. It is further affirmed, by Jilek (p. 57) for Pola, by Willkom for the plateau of New Castile (Spain) and Valencia, by numerous authorities for Tennessee, Virginia, Pennsylvania, and other of the Central States of the Union, and for Belgium by Meynne, who remarks (p. 226): "During hot summers the fever acquires more intensity and greater extent than in ordinary years. . . . We may, indeed, establish a general rule that the intensity of the paludal miasm is regulated by the intensity of the heat." Still further, by Goldschmidt and many other writers for the malarious localities on the northern coast of Germany, by Mondineau (p. 13) for Landes, by Tessier for the Sologne, and by Godelier for Charente. So

that when Dutroulau, in summing up all these facts, says (p. 165): "The steady rise of the thermometric mean in warm and palustral climates must be regarded as unquestionably a more potent aiding cause of endemic fever than the sudden rise of the thermometer in certain localities"—he expresses not only the view of observers in tropical and sub-tropical regions, but also the purport of the observations that have been made in higher latitudes. As to the coincidence of great epidemics or pandemics of malaria with remarkably hot years, there are likewise many observations from the most diverse parts of the globe; among others, for the extensive and severe epidemics of 1809—1811 in Mysore, and of 1829 and 1841 in the North-West Provinces, the epidemic of 1853 in Amboina, where, according to the account of Popp (p. 14), the pestilence appeared with calms and a very high state of the thermometer following heavy rains, ("in die mate, dat de oudste zich met herinnerden, eene dergelijke saisonsverandering te hebben bijgewoond,") the epidemic of 1829-30 in Brazil (Sigaud, p. 170), many severe epidemics in the Netherlands and Germany (Alkmaar in 1556, Friesland in 1748, Amsterdam in 1834, Bruges in 1842, and the general prevalence of the sickness in 1719, 1807, 1811, 1826, 1846-47, and 1855), the epidemic in England in 1657 (Willis), that of 1831 in Sweden, and that of 1834 in Denmark (Bremer).

But although there can be no reason to question the general importance of high temperature for the production of malaria, there is just as certainly another series of facts which serves to keep that significance within due limits. On the one hand we meet with the fact, noteworthy in this connexion, that, in higher latitudes, the malarial fevers which have prevailed endemically or epidemically in spring undergo for the most part a considerable remission on the setting in of summer heat, that they do not revive until the cooler weather of autumn, and that the disease has often attained a wide diffusion in spring during remarkably raw and cold weather (as at Halle in 1701, in Dalecarlia in 1772, Copenhagen in 1724, Lüneburg in 1797, Riga in 1799, Baireuth in 1812, Würzburg in 1824, and various places in North Germany in 1847). It is this that has led Huss to conclude from his Swedish experiences (p. 83): "en låug, kylig og fuktig vår alstrar städse et större antal frossfebrar, än en kort, mild och torr." Next there is a circumstance not to be left out of sight, that, in the regions of severe malaria, the disease shows itself and attains a wide diffusion, not at the height of summer, but only when the high temperature is declining in late summer and in autumn, and,

for the tropics in particular, at the end of the hot season. And, as many observers state, this is directly due to the great diurnal range of the temperature that occurs at that season.

Twining (II, p. 207) expresses himself very decidedly on this point, basing on his experience in Bengal: "The frequency of intermittents is augmented beyond all proportion after the cold nights and foggy mornings commence, and when the heat of the day, though much decreased, is followed by a greater degree of depression of the thermometer during the night than happens at any other season of the year;" and the same opinion is stated by Geddes (II, p. 163) for Madras, by Nicoll and Day ('Indian Annals of Med.,' 1859, January, p. 88) for the Deccan, by Hamilton for Honduras, by Cambray, Worms, Phillippe, and others for Algiers, by Aubert-Roche for the Arabian coast, by Forchi and Fourcault for Rome, by Faure (p. 47) for Greece, and by Kaputschinsky, Popoff, Sachs, and others for Southern Russia. Nepple, whose experiences relate chiefly to the Bresse country, says (p. 135): "It is an indisputable fact that this malady does not prevail equally at all seasons; that it is not until the end of summer, or, in other words, during the period which directly follows the great heats, that a large number of persons are attacked at once," adding, at the same time, that the extent and severity of the sickness is in direct proportion to the high degree of heat in the preceding summer. Severe epidemics of autumnal fever, in somewhat cool weather, have been observed in 1615 and 1684 in various parts of Germany, in 1657 in England, in 1724 and 1726 in Paris, in 1764 in the Bresse country, and in 1835 in Vienna.

It is further of some account for the question before us that the endemic and epidemic malaria of the tropics disappears on the setting in of the so-called cold season, or, in other words, that the disease occurs there only sporadically with a temperature at which, in higher latitudes, malaria still continues in full force and potency. Finally, there is the fact that in regions with a temperate climate, and even with a climate reckoned as cold, not only has an epidemic which began in the autumn, continued through the winter, but also that epidemics of malaria have even developed under a winter temperature.

Frank[1] observes: "I have several times seen at Wilna intermittent fever showing itself in the month of February when the thermometer showed 20° R. of frost and even more;" in Kasan, as Blosfeld tells us, an epidemic of malarial fever occurred in the winter of 1841-42 in the

[1] 'Prax. med. univ. præcepta,' [De febre intermitt., § xxvii, 9 Lips., 1826, i, p. 262.

midst of severe cold; Meyersohn (p. 259) writes from Astrakhan: "It is a fact that the fever was prevalent even when the thermometer showed twenty degrees of frost and more;" and Walter (p. 99), speaking of Kieff, says: "In our eastern governments [of Russia] the epidemics of fever show themselves even when the whole country lies under a firm covering of ice; and, if the statements of physicians, which I have had the opportunity of hearing, are correct, they reach a higher intensity than in the hot summer time. That ten or fifteen or twenty degrees of frost does not with us ward off fever, is proved by the scarcity of quinine, which is much felt in the months of December and January, if the supply of the drug have been used up in the course of the year."

If we would rightly state these etiological questions, and correctly interpret the answers to them, we should, in my opinion, agree to the declaration of Jacquot:[1] "if the rise of temperature cannot of itself create the fevers, it appears as if it were capable of increasing their frequency and gravity;" while we must at the same time recognise the fact that the relations of malarial production to influences of temperature are by no means so simple as we have been accustomed to deem them.

§ 67. Influence of Moisture—Rain or Dew.

An influence on the development of the disease not less pronounced than that of the temperature is exerted by the degree of *atmospheric moisture*, or of the *atmospheric precipitations* that result therefrom. Usually this influence is manifested in the occurrence of malaria, or in an increase in the amount of sickness, after copious rains, especially if they be followed by dry weather and a high temperature. But inasmuch as this etiological factor really deals with the saturation of the soil caused by the precipitations (rain and dew), and with the fact to be afterwards proved that the saturation, when complete, sets limits to the development of the malaria, then it is self-evident that we are always concerned here with a certain measure, or with the relative amount of the precipitations. This mean or measure will stand in a definite ratio to the condition of the soil; the fall

[1] 'Gaz. méd. de Paris,' 1848, p. 589.

must be all the more copious, if it is to aid in the production of the disease, when the soil is naturally dry, while, on the other hand, a very heavy fall on a naturally wet soil will prevent the development of the disease till such time at least as the soil has again become in a measure dried through the evaporation or sinking down of the moisture within it. This law, deduced from a long series of observations, is on the whole well borne out by circumstances; but here again there are a certain number of prominent facts that do not suit themselves to it,—a good many contradictions of which the explanation is still to seek.

In the malarious regions of the tropics the fevers appear, as a rule, at the beginning of the rainy season, they increase in extent and severity with the increasing rain-fall,[1] remit usually at the height of the rains, especially if they be very heavy,[2] and reappear towards their cessation or directly after the rainy season, which is, as a whole, the season when the conditions are most unfavorable to health. Showers of rain, also, in the hot season, and all alternations between drought and moisture, are usually followed by an exacerbation of the endemic;[3] and one may often detect a direct relation between the extent and severity of the endemic or epidemic and the degree of the preceding rainfall.

"Increased moisture leads to increased admissions for fever," says Day,[4] speaking of the Deccan, and opinions of the same purport are given by Annesley (p. 523) for Bengal, Geddes (p. 94) for Madras, Griesinger (after Penay) for Khartoum, Aubert-Roche for the Arabian coast, and by others. Side by side with these accounts from the tropics, of severe malarial epidemics after continuous rain, there are communications by Langstaff for Delhi in 1829, McGregor for the plain between Delhi and Karnaul in 1841, Spencer for Moradabad in 1836, Geddes (I) for Kaddapah in 1826, and by Sigaud for Brazil in 1829-30. To the same category, also, belong the reports of severe epidemics in Honduras in 1861,[5] and Ramandrag in 1863.[6]

[1] Annesley, p. 520, Pritchett, p. 108, Bernouilli, Day (V), p. 240, Griesinger, p. 374, v. Frantzius, p. 319, Vaillant, p. 12.

[2] Pruner, Meller, Forbes, v. Frantzius.

[3] Geddes (II), p. 136, Day, l. c., v. Frantzius. Blanc observes that in Massowah [Red Sea] the smallest precipitations suffice to call up the malaria.

[4] 'Indian Annals,' 1858, Jan., p. 71.

[5] 'British Army Reports,' 1861, p. 73.

[6] 'Madras Quart. Journ. of Med. Sc.,' 1863, July, p. 118.

The facts come out practically the same in subtropical regions and in higher latitudes. There also we find:

1. Malarial fever appearing as an endemic or epidemic either when the rains set in after a long period of heat and drought, or, again, when the rains cease and give place to warm and dry weather.

Thus Link relates that when the first rain fell in Athens in the beginning of October, 1838, after a summer that had been very hot and dry all through Greece, every hospital in the city became filled with the sick all at once. Under the same or at least similar circumstances, malarial epidemics sprang up at Lucca in 1648, at Rome in 1795, at Olmütz in 1783, in Walcheren in 1809, at Zevio in 1811, at Rimini and La Rochelle in 1827, in Istria in 1833, at Grenada (Haut Garonne) in 1845, at Emden, at Breslau, and in Galicia in 1846, at St. Maure (Indre-et-Loire) in 1848, and at Fürth in 1859. With regard to the disease increasing as the rains cease and dry and warm weather sets in, Hippocrates had already said:[1] "If the winter be of a dry and northerly character and the spring rainy and southerly, there will necessarily be acute fevers."

We meet with numerous epidemiological experiences corresponding to this; thus, the same sequence has been observed in North Carolina, according to Dickson,[2] at Fort Merrill, Texas (Moses),[3] in Algiers (Jacquot[3] and Bachon), on the Siberian Steppes (Woskesensky and Rex), and in the Danubian Principalities (Leconte).[4]

2. The endemic or epidemic dies out at the height of the rains if they are abundant.

In Sardinia, says Moris, the endemic attains its acme in autumn whenever the first rains set in, and begins to decline only when the plains are entirely under water; this phenomenon came out very markedly, according to Dutton, in the epidemics of 1823 and 1824 in Delaware County, Pennsylvania.

3. The disease is common in wet years and of rarer occurrence in dry years.

With reference to the prevalence of malarial fevers in the Southern and Western States of America, Cooke (I) says: "Wet summers are sickly and dry summers are healthy," adding the noteworthy remark: "except in the neighbourhood of marshes, ponds, and rivers," and he at the same time calls attention to the enormous diffusion of the disease

[1] Aphorism. iii, § 11 (Ed. Littré iv, p. 490, *ἢν μὲν ὁ χειμὼν αὐχμηρὸς καὶ βόρειος γένηται, τὸ δὲ ἔαρ ὑπόμβριον καὶ νότιον, ἀνάγκη, τοῦ Θέρεος πυρετοὺς ὀξέας*).

[2] 'U. S. Army Reports,' 1856, p. 353.

[3] (II), p. 751.

[4] P. 20.

in Pennsylvania, Maryland, Virginia, Ohio, Mississippi, and Alabama in the very rainy year of 1823. Sutphen expresses a similar opinion for the State of Michigan, Williamson for North Carolina, Meynne[1] for Belgium (with special reference to the wet year of 1859 when there was much fever as compared with the dry year of 1858), Schröder[2] for South Bavaria, Bailly[3] and Colin[4] for Rome, Ely for Paestum, Troussart and Gaucher (II) for Algiers, and Rafalowitsch for Syria. For certain localities this ratio between the quantity of the rainfall and the amount of sickness is proved by statistics; for example, the following table is given by Jilek for Pola.

	1864.	1863.	1866.	1865.	1867.	1868.
Rainfall, in Paris inches .	18·44	14·25	12·10	3·44	5·49	1·5
Cases of fever, per 100 men	51·4	48·6	36·3	35·4	22·9	14·2

The apparently contradictory results for 1865 and 1867 are explained on the one hand by anomalous conditions of temperature, and also by the great differences, as regards malaria production, between moderate falls of rain in rapid succession and the same distributed over longer periods. Armand[5] remarks that in the year 1843, in which there was hardly any rain in Algiers from May to October, the number of admissions for malarial fever among the French troops was 52 per cent. of the effective force, while in the previous rainy year it was 71 per cent.

According to Pendelton,[6] the ratio of sickness to rainfall in the malarial fevers of Central Georgia may be represented as follows:

	1845.	1846.	1847.	1848.	1849.
Rainfall in Spring and Summer[7] .	0·61	1·37	1·47	1·13	1·50
Rainfall in Autumn	0·86	0·64	0·45	0·75	0·07
Amount of sickness[8] . . .	16·28	48·10	41·80	29·00	32·48

[1] P. 336. [2] P. 100. [3] P. 127.

[4] (II), p. 75. "It is a general rule," he says, "that the more rainy the year has been, previous to the breaking out of the fevers, the graver and more numerous these are; while, on the other hand, there is a minimum of sickness in dry years, even if they be very warm, as we found in 1865."

[5] (I), p. 132.

[6] 'Southern Med. Reports,' i, p. 327.

[7] The figures give the amount of rain as calculated with relation to the mean rainfall = 1.

[8] Proportion of malarial sickness per hundred of all cases of disease observed by the author in the same period.

But there are still, as we have said, many unexplained exceptions to the rule, which serve to show how far we are at present from having a complete insight into this particular factor. In the years from 1868 to 1872, an enormous increase of endemic malarial fever was remarked in India; this striking phenomenon was particularly obvious in Madras, where the mortality from malarial fevers was:

In 1868 . . .	105,692
„ 1869 . . .	132,346
„ 1870 . . .	151,027
„ 1871 . . .	193,398

But the years 1868 and 1871 were notable for the almost complete absence of the north-east monsoon, and still an increase in the amount of sickness was uniformly remarked all over the Presidency;[1] in 1872 abundant rains appeared again, but in that year a very noteworthy decrease in the amount of sickness took place. In the official accounts of the state of health in India in the years 1872 and 1873, it is explicitly stated that there, as well as in other regions of the tropics, wet years have very often been distinguished by a highly satisfactory state of health, and dry years by very unfavorable sanitary conditions. Thus, in the year 1869, there was a widespread epidemic of malaria in the Punjaub, the North West Provinces, and down to the mouth of the Indus; and, as Cunningham points out,[2] the one factor that could *not* be held responsible for the outbreak of the disease over that great tract of country, was the saturation of the soil. Another fact in this connexion is noted by Wenzel;[3] on comparing the rainfall for twelve years, while the harbour works in the Bay of Jahde were going on, with the corresponding annual amount of malarial disease, he found that the popular opinion prevalent there, that want of rain and great drought favoured the rise and diffusion of malaria, was no more applicable to that district than the converse view, and "that a saturated state of the ground, and the exact opposite, were almost indifferently concerned in the production of marsh malaria." Thus, he observed:

[1] Account in the 'Madras Monthly Journ. of Med.,' 1872, v, p. 298.

[2] 'Tenth Annual Report of the Sanitary Commissioner with the Government of India,' 1873. Calcutta, 1874.

[3] P. 146.

In three years (1858, 1859, and 1868), severe epidemics with a rainfall of 2″ to 4″ below the annual mean.

In one year (1861) a severe epidemic with an excessive rainfall, 5·25″ above the mean.

In two years (1862 and 1863) severe epidemics with a rainfall that diverged from the mean scarcely at all.

In 1865 a slight epidemic with a deficiency of more than 6″, and in 1866 a second slight epidemic with an excess of 2″ of rain.

On two occasions, with a great excess of rain in all the four seasons of the year (1860 and 1867), and on one occasion, with a great deficiency (1864), there was no endemic prevalence of the disease whatsoever. If we take into account all the other allied meteorological factors, such as temperature, clouds, and the like upon which the evaporation depends, we may still safely conclude with Wenzel "that the production of malaria in a marsh is neither unconditionally furthered, nor unconditionally hindered by the saturation or by the dryness of the ground, nor indeed influenced in any uniform and regular way." The fact that epidemics of malaria coincide in general somewhat more frequently with drought than with abundant rainfall, depends less, as Wenzel adds, upon hydro-meteorological than upon other weather conditions, particularly the temperature, which is lower on an average in wet years than in dry.[1]

§ 68. Influence of Winds.

In considering the question of the influence which *winds* exert on the occurrence and diffusion of malaria, we have to take into account, on the one hand, the property of the air in motion as a carrier of material substances, or of the morbid poison, and, on the other hand, its modifying effect upon the statical conditions of atmospheric heat and

[1] In Dittmarschen also, as Dose points out, severe endemics coincide with dry years, and the milder endemics with wet; but here the difference is much more pronounced than in the Jahde district. Thus in the wet years of 1842-46, 1849-51, 1856, and 1863, there occurred in all 1242 cases of malarial sickness, or a yearly average of 124·2; while in the dry years of 1847-48, 1852-55, and 1857-62, a total of 5614 cases were observed, or a yearly average of 467·8.

moisture. The first mentioned relation of the wind to the diffusion of the disease will be treated of subsequently. With regard to the other, it has been shown by several observers, Koreff, Salvagnoli-Marchetti, Guislain, and others, that the fever in Sicily and on the mainland of Italy appears to increase both in severity and in amount during the prevalence of the African desert-wind or sirocco. It is an open question how far the "variations in the electrical tension of the air" caused by the sirocco come into the problem, as Marchetti contends that they do; it is, at any rate, a less remote consideration to take into account, as pathogenic factors, the rise of temperature, and above all the increased amount of atmospheric moisture brought by that wind. The great importance, for the development of malaria on a suitable soil, of the comparatively small precipitation caused by a heavy dew-fall, is clearly proved by the occurrence of the disease under those circumstances in districts where there is no rain, particularly on the coast of Peru.

§ 69. Altitude and Configuration of the Ground.

The pronounced endemic character of malarial disease throughout so many large regions of the globe differing widely from one another in a great variety of circumstances, meteorological, anthropological, and social, justifies us in assuming at the outset that the occurrence of the disease is associated with certain conditions of the locality, and especially with *conditions of the soil*. There are, indeed, few points in the etiology upon which observers are so agreed, as that the soil has an influence in the production of malaria. But, as regards the actual pathogenic importance attaching to factors situated in the soil, as well as the way in which these factors help in the production of malaria, there are still wide differences between the views of the various investigators; and it is only through a careful analysis of the facts that we shall succeed in letting some light in upon this often explored, but still very obscure region of etiology.

As regards the relation of the *altitude* and *configuration of*

the ground to the endemic and epidemic occurrence of malaria, the general law may be expressed by the formula, that the extent and severity of malarial diseases diminish in proportion as we ascend above the sea level. This relation is most marked in the diffusion of the disease in mountainous regions; but it comes out unmistakeably also in a hilly country or among downs, as well as on a moderate declivity, or even on a level plateau with basin-like or cup-like depressions. It is always, *ceteris paribus*, the deepest points that are affected most by the disease; the state of health is the more favorable the higher the locality.

This fact in the distribution of the disease is illustrated on a large scale in the valley of the Mississippi and upon its western declivity; "the constantly increasing elevation of the desert to the west of the Mississippi," says Drake,[1] "is, no doubt, one cause of the disappearance of the fever under the same parallels in which it prevails on the banks of that river." We meet with the same circumstances farther south on the slopes of the Rocky Mountains (Andes) of Texas (*Bracht, Meyer*), Mexico (*Jourdanet, Liberman*), Central America (*Bernhard, v. Frantzius*) and South America; also on the slopes of the Atlas in Algiers (*Finot, Philippe*), the table-land of Abyssinia (*Harris, Courbon*), the elevated plain of Armenia (*Wagner*), the South German Alps, and the Apennines. Concerning the last-mentioned mountain chain within the (quondam) Kingdom of Naples, it is stated by Dorotea[2] that malarial fever does not occur at all in the Alpine regions, *i.e.* at a height of 700 toises (1400 metres) and upwards, that in the Montana or cultivated zone (350 toises and upwards) only the slight and simple fevers are observed in small numbers, while in the zone of the foot-hills the sickness begins to show itself more frequently, and the coast region forms its proper territory. To the same effect is the opinion of Salvagnoli-Marchetti on the distribution of the disease in Tuscan territory; in the mountainous and hilly country on the one hand, and the Maremma on the other. Even in the regions of most intense malaria, this influence of the elevation of the soil is unmistakeable. Thus, at many points on the West Coast of Africa, the disease becomes

[1] I, p. 715. [2] P. 181.

rarer and milder the farther one goes from the level of the coast and the higher one rises (*Oldfield, Ritchie, Hugiot*); the heights surrounding Bussorah are almost free from malaria, whilst Bussorah itself is infested by fevers of the worst kind (Hyslop); there is immunity also on the moderately elevated plateau of the Southern States of America. On a smaller scale this influence is shown in the endemic prevalence of malarial fevers, often within narrow limits, in low grounds, as in the central depression of the country in Sweden, especially round the Lakes Malar and Wener, in the English county of Gloucester (*Nash*), in the district of Medoc (*Le Gendre*, p. 13), and at innumerable points in the south-west of Germany.[1] It is a universally recognised fact in malarial epidemics that, in districts with an undulating surface, the deepest localities or portions of localities are attacked first and most severely.

It stands to reason that the immunity of elevated regions from malarial disease is only a relative one, and this is explained by a consideration of the conditions upon which it depends. On the one hand, the mean summer temperature is a determining factor; the height to which malarial fever ascends at the various high-lying points on the globe is in definite ratio to the geographical position, and accordingly in an inverse ratio to the latitude of the place.

In the alpine parts of Germany the limit of malarial fever may be placed at an elevation of from 400 to 500 metres, in Italy it rises to 600 to 1000 metres, and to much the same height in the mountainous parts of Corsica (*Gouraud*, p. 29), on the slopes of the Atlas in Algiers (*Leclerc, Armand* (I) p. 125), and in the elevated mountain valleys of the Lebanon (*Pruner*). At still higher elevations (up to 2000 metres) we find malarial fever endemic on the slopes of the Himalaya and the high table-land of Ceylon (in the former at Kussuli (Simla),[2] and in the latter, at the same altitude, in Newera Ellia[3]), and in the form of "mountain fever" on the eastern slopes of the Rocky Mountains.[4] In the Peruvian Andes the fever is met with at elevations even up to

[1] See the data given by Schröder as to the distribution of malaria in South Bavaria.

[2] Ireland, p. 21.

[3] This statement, resting on the authority of Marshall and Cameron, p. 71, has lately been confirmed by Massy (l. c., p. 497).

[4] Ewing, Bartholow, Waggoner. See also the accounts given by Milhan and Brewer for Utah Territory in the 'U. S. Army Reports,' 1860, p. 304 ff.

2500 metres or more, as in Tacna, according to Hamilton, and along the mountain road as far as Arequipa.

The second factor in this relative immunity of elevated localities from malaria is undoubtedly the state of the soil as regards moisture, which is naturally different from the degree of moisture in the plain. I shall recur to this circumstance in the sequel; I will only mention here, and merely by way of illustration, that wherever malarial fever is endemic at more or less considerable elevations, the seat of the disease is always a valley with a small declivity, or a basin-like depression in a plateau, while the open levels, except so much of them as lie immediately at the foot of shelving mountain spurs, are, like the mountain ranges themselves, for the most part exempt.[1]

§ 70. Geological and Physical Characters of the Malarious Soil.

The most important aspect of the origin of malaria is undoubtedly that which presents itself in the question how far the disease depends, as an endemic or epidemic, upon the *geological* and *physical conditions of the soil*, upon the kind of rock, the porosity, the degree of saturation, the amount of organic detritus, and, further, upon the tillage, and perhaps, also, upon the products resulting from cultivation.

Whether *geological characteristics of the soil* exert an influence on the production of malaria is, to say the least, questionable; at all events the opinions that have been put forward on that subject have not been accepted.

Thus, Heine has been led to lay particular stress, from the pathogenetic point of view, upon the amount of iron in the soil in the hilly fever-regions of the Deccan. On the other side, McClelland (p. 120) has shown that the laterite, to which Heine's suggestion refers, possesses all those physical characters which are peculiar to malarious soil in general; and, conversely, we find the same formation of

[1] The facts published by Steifensand, p. 115, on the prevalence of malarial fever in the basin-like depressions among the mountains of the Lower Rhine are very instructing, on a small scale, for estimating these conditions.

soil in many other districts adjoining the malarious localities, which are themselves free from malaria, such as Midnapur,[1] Chota Nagpur, and Dorunda,[2] as well as Simla and other points on the foot-hills of the Himalaya.[3] In like manner we must reject, or at least receive with much caution, the notion, which I myself long shared, that a large quantity of salt in the soil, especially common salt and saltpetre, conduces materially to malarial fever; this opinion is chiefly based upon the endemic prevalence of the disease on the west coast of Italy, on the table-land of New Castile, the steppes of Russia, the prairies of North America, &c.; but there are other regions, such as the pampas of the River Plate States, which enjoy a remarkable exemption from malaria, although their soil contains the same ingredients.

We may, in the meantime, safely limit the influence of the soil in the production of the malaria to its distinctive *physical characters*, among which the conformation and the kind of rock are certainly of real importance. But we discover, farther, that no formation and no kind of rock absolutely excludes the occurrence of malaria. It can be shown that the intensity of the morbific influence is materially increased by the porosity and hygroscopic character of the soil; and accordingly the alluvial and diluvial formations are classical ground for malaria, while the older formations are more or less exempt in proportion to the compactness of the rock.

The chief seat of the endemic malaria of the alluvium or diluvium is always found where there is a permeable and highly hygroscopic *clay soil* (clay, loam, clayey marl, marsh, &c.); a porous *chalk soil* is less favorable to it, and least favorable of all is a *sandy soil*. The relative exemption from the disease of the last mentioned kind of soil may be explained by reference to its distinctive physical characters; readily open to saturation in consequence of its loose structure, it is unable to retain the absorbed moisture, and it becomes dry almost as soon as it is saturated. It is only when a soil of porous chalk or a sandy soil rests upon highly hygroscopic clay, or a firm kind of rock, that the former are more apt to be associated with malaria; the substratum is little adapted for conducting water, and the moisture that

[1] Goodeve (II). [2] Dunbar. [3] Ireland.

has sunk into the chalk or sand is unable to escape from them at once, and so this saturation is maintained for a considerable time.

Linnæus was the first, as far as I know, who advanced the notion that malarial fevers were frequent on clay soil, on the strength of his observations in Sweden. The same fact has lately been demonstrated very conclusively by Meynne (p. 302) for Belgium, and we find it to hold good for almost all the great malarious regions, and for the smaller spots of malaria no less, such as the marshy districts of England (Cambridgeshire, Lincolnshire, and Gloucestershire[1]) and the department of Calvados.[2] The great liability to malarial fever of a clay soil, as contrasted with the exemption of a sandy soil, comes out with especial prominence in all the epidemics that have occurred in the malarious regions of the North German plain and of the Netherlands.

The exemption from malaria enjoyed by the islands of the West Indies with a chalk soil, such as Barbadoes, is very remarkable when contrasted with the special prevalence of the disease in the islands of volcanic formation.[3] In the sketch which Jourdanet (p. 150) gives of the malarious regions of Mexico, we read: "In the towns of Campeché and Merida, both built upon chalk soil, intermittent fevers are not very common in comparison with other places," and this statement is confirmed by Debout as far as relates to Campeché.

Concerning the distribution of malaria in Belgium, Meynne states (p. 307): "It may be said in general that the strata of sand, permeable and containing very little of foreign substances [viz. clay], are eminently healthy." This exemption of sandy soil from malaria comes out prominently, as we have said, in the epidemic fever of the coast of Germany and the Netherlands; thus Fricke, in his report upon the epidemics of 1826 and 1827, states (p. 49): "This form of disease showed itself in all localities which had the so-called clay soil; from places with a sandy soil adjoining them, it was as if cut off, although the latter were likewise affected

[1] Royston, Nash.

[2] Account in the 'Lond. Med. and Phys. Journ.,' lxvi, p. 87.

[3] 'British Army Reports (West Indies),' 1838, pp. 26, 27.

by the inundations." Further evidence of this occurs in the exemption of the sandy parts of Calvados as contrasted with the districts of that department that rest on clay; McClelland (p. 123) also states that the occasional spots, where there are interruptions of light sandy soil in the highly malarious laterite (clay with iron) running through the South of Hindostan from Midnapur to Sumbulpur, are not subject to the endemic disease notwithstanding their immediate contiguity to the most intense malarious foci; Lord says that in Lower Sind, which is notorious for its bad forms of malarial fever, the disease is rarely met with on a sandy soil; and, lastly, it is observed by Annesley that the relatively favorable conditions of health at many points on the Malabar coast (Madras Presidency) are explained virtually by the sandy soil of the localities.

In complete agreement with the circumstances here spoken of, we have, finally, the fact that in localities which rest upon a rocky bottom, but are still the seat of endemic malarial fever, there is always a more or less thick layer of permeable alluvium, or diluvium, or mineral detritus spread over the firm rock, and always, therefore, a hygroscopic upper soil.

We meet with malarial fevers under such circumstances on the rocky soil of Guernsey (*Hoskins*), in Gibraltar, and in certain parts of Tennessee and Kentucky (*Drake*); further, on the greater part of the west coast of Italy, from the Tuscan Maremma down to Sicily, where there is, resting upon the firm volcanic rock (basalt), a deposit of clayey marl of various thickness mixed with broken rock, and upon that again a stratum of the newest alluvium rich in salt (salmastraje); again, in the Canary Islands (*Lopez de Lima*), where basalt or trachyte has volcanic tufa or clay resting upon it; in Bellary on the so-called "black cotton ground,"[1] which is basalt covered with volcanic detritus; in Malwa, on trap with an upper stratum of sand (*Ranken*), and in Cutch (*Winchester*); and, finally, in the basins of the Tempisque and Rio Grande in Costa Rica, where, according to v. Frantzius (p. 318), there is also volcanic tufa deposited on firm rock.

[1] Day, 'Indian Annals of Med.,' 1859, Jan., p. 86.

§ 71. Saturation of the Soil.

Under all these circumstances, then, the peculiar malaria-producing property of the soil seems to depend upon copious *saturation of the ground* and upon the concurrent formation of organic vegetable detritus. This saturation may be brought about by:

(1) Atmospheric precipitations, the importance of which for the production of malaria has already been dwelt upon, while it has been at the same time proved that the intensity and amount of the disease not unfrequently stand in a direct ratio to their amount.

(2) Nearness to larger or smaller basins, whose enclosing line is sufficiently low to prevent the drainage setting towards the more elevated rivers, lakes or pools; so that the soil is constantly saturated, and the sub-soil water is high or low according to the amount flowing in.

(3) Inundations occurring periodically or at irregular intervals.

(4) Saturation of the ground with sub-soil water, a circumstance which is calculated to throw light upon the occurrence of malarial diseases in many localities situated remote from river basins, and whose soil cannot become saturated in other ways.

The production of malaria takes place on the largest scale under (2) and (3) of the above-mentioned conditions. Firstly, on the low coasts of tropical and subtropical countries, and on the damp, alluvial, and often flooded banks of their great rivers; and in higher latitudes also, in many great foci of disease where the same peculiarities of the soil are recognisable, for example, the shores of the Caspian, the lower basin of the Volga, of the Danube, of the Rhine, of the Elbe, and of the Vistula. In the second place we find malaria exceedingly common in small and often definitely circumscribed spots by the sides of lakes, small streams or brooks, pools, ponds, and ditches, and extending just as far as the basin makes its influence felt in saturating the soil of the neighbourhood. There can be no well-founded doubt, therefore, about the intimate causal connexion between a wet

soil and the production of the disease ; and the former may be regarded as characteristic to some extent of malarious localities. A very striking example of this is furnished by the hygienic conditions in the Gironde, of which the following account is given by Gintrac : The department is divided by the Garonne into two almost equal parts, a north-eastern with higher elevation and a thoroughly dry soil, and a damp south-western division, the southern extension of which is the plain rising somewhat towards Landes, with the highly malarious Bordeaux at the northern apex of the triangle. Of 484 patients admitted for malarial fever into the Bordeaux hospital during four years, 105 came from the arrondissements on the eastern bank of the Gironde, and 379 from those on the western ; but, inasmuch as the population of the first division was 254,150 and that of the second only 179,429, the ratio of the sickness in the whole population was in the former case 1 in 2420 and in the latter 1 in 473.

Besides these malarious regions, we have, thirdly, the endemic occurrence of the fever in localities that are periodically inundated by irrigation for the purposes of agriculture. A classical example of this is found in the large malarious districts of countries where rice is much cultivated, as, for example, in many parts of India, especially the western division of Khandeish (*Williamson* I), in Java, in Ceylon (*Cameron*, p. 71), in Hong Kong (*Wilson* (I), p. 147, *Dill*), and other parts of the south-west of China, in Japan (*Wernich*), in Greece, in the rice-growing districts of Upper Italy (*Ferrario, Savio, Maffoni*), Sicily, and the north of France, in Portugal, and, in the Western Hemisphere, at Savannah (Georgia), where, according to Daniell (I) malarial fever has become endemic since the rice-fields were laid out, and round about Charleston (*Simons*, p. 406), and at other places in the Southern States. In like manner, we may account for the prevalence of the disease in localities with extensive meadows and much cultivation of hemp (as in the low grounds of the provinces of Pinerolo, Saluzzo, Turin, Ivrea, and parts of the provinces of Asti and Alessandria in Upper Italy), in the mountain valleys of Syria in consequence of much garden-irrigation (*Richardson*), on the gold-fields of California, where the ground, originally dry, has

been put under water for the purpose of gold-digging and agriculture (*Logan*), and under other conditions of a like kind.

Fourthly, and lastly, the importance of a wet soil for generating malaria is especially well shown in connexion with the endemic occurrence of malarial fevers in localities which, although remote from a river basin, have their soil abundantly saturated by subterraneous springs. Van Swieten was the first, and after him Pringle and Monro, to direct attention to this fact, and further inquiries in the various localities have afforded an interesting explanation of it. There are in Sicily, as Irvine (p. 5) states, many beds of streams which are quite dry in summer (the so-called *fiumari*), and malaria is endemic in their neighbourhood; inquiry has shown that, in the bed of the stream higher up, there is a small rill of water which appears to sink suddenly into the sand, while in fact it pursues its way underneath the channel. This applies, for example, to the large *fiumare* running to the north of Messina, which appears to be quite dry in summer, but rapidly fills with fresh water if one digs not more than a foot or two; "I have often observed," says Irvine, "that such fiumares as have, amongst the natives, the reputation of being subject to malaria, have streams of water running all the year in their superior parts." The case is probably the same with those seemingly dry malarious places in Sardinia, where the quivering of the ground under the foot (hence called "tremulo" by the natives) betrays the presence of sub-soil water. A further contribution to this subject is made by Celle (p. 10) with reference to the endemic occurrence of malarial fever at several places in North Africa: "There are" he says, "collections of subterraneous water, arising either from hidden sources, or owing their origin to the infiltration of rain water; these collections rest always upon impermeable strata of argilaceous rock or marl, so that they have no other way of getting reduced except by the prolonged action of the sun on the surface. Not to mention the Sologne, there are certain parts of Algiers, of the coast of Tripolis and of Darfour, that are subject to the effects of these hidden deposits." Precisely the same explanation may be resorted to for the occurrence

of malaria in several rainless oases of the Sahara, whose geological formation must be represented in some such way as follows: Basin-like depressions, of various extent, in a rocky or highly hygroscopic bottom, form reservoirs and channels for the collection of subterraneous water; they are covered by a layer of alluvium, the surface soil of the oasis, and they swell in volume in spring in consequence of the snows melting on the interior mountain ranges of Central Africa. The influence of these subterranean collections of water upon the saturation of the soil above them is so great that, even in the intervals between the various oases, the sandy surface changes in spring into almost green meadows, affording a periodical sustenance to the cattle of the nomadic population of those regions. At several places, in Spain and Greece also, malaria is found, according to Armieux,[1] under the same circumstances.

§ 72. Organic Matters in the Soil.

The *amount of organic matter in the ground* is the last of those properties of the soil which we have found to stand in a causal relation to the origin of malaria. Observers at the most diverse points on the globe are nearly unanimous in saying that the development of the malarial poison depends directly or indirectly upon the processes of decomposition of organic and particularly of vegetable matters in or upon the soil, and that it is in a measure bound up with those processes. Drake (I, p. 709) sums up the American experience on this point with the words: "It is a safe generalisation to affirm that, all other circumstances being equal, autumnal fever prevails most where the amount of organic matter is greatest, and least where it is least." We shall consider afterwards how far this opinion applies generally.

§ 73. Changes in the Soil—Cultivation, Neglect of Cultivation, Excavations, Volcanic Disturbances.

The experience of variations in the amount of sickness coincident with changes in the condition of the soil is

[1] 'Gaz. des hôpit.,' 1865, Sept.

important as enabling us to estimate the influence of the soil on the production of malaria. There are here three main considerations:

1. The decline or even the complete extinction of the endemic after the *drying up of a previously damp or marshy soil*, a fact which has been conclusively proved by observation hundreds of times in all parts of the world.[1]

2. The fact that, *when the water is high and the ground completely covered by it*, the endemic or epidemic disappears, fresh cases of the disease appearing only after the water has run off and the surface of the ground has been laid bare. Classical examples of this are furnished by the malarious regions periodically inundated on the banks of the Nile, Indus, Euphrates, Ganges, Senegal, Niger, Mississippi, and other rivers, where the endemic always begins after the waters have begun to subside. There is another illustration of the fact in those rice-fields in India which are always under water, and, as Annesley remarks, are the least dangerous to health. Experience also in Turkey (*Sandwith*), in Sardinia (*Moris*), at several places in the Southern States of the Union (*Nott*), in England (*Royston*) and elsewhere, proves that the complete flooding of marshy ground, and the filling up of standing pools, ditches, and the like causes the endemic to disappear as certainly as if they had been dried up.

3. The way *in which the soil is treated* has an influence upon the amount of sickness. Thus the breaking up of virgin soil and other operations of that kind, the cutting down of woods, and the neglect to cultivate ground that used to be tilled, are favorable to the occurrence and prevalence of malarial disease; while, on the other hand, a

[1] Meynne (p. 286) writes thus of the improvement in the sanitary conditions of Belgium: "The progressive diminution of endemic intermittents in the coast belt is an ascertained fact, and that diminution has everywhere coincided with the disappearance of marshes, the extension of cultivation, and the establishment of a regular system of storing water." The same applies equally to many localities where these principles are carried out on a large or small scale, particularly to the notorious malarious districts of France, the Sologne, the Dordogne (*Sclafer*), and the like, where there has been a considerable decline in the sickness since the amelioration of the soil was undertaken; many districts in them, which used to be almost uninhabitable on account of malaria, now enjoy the most favorable sanitary conditions.

careful and regular cultivation of the soil contributes materially to improve the health of a locality.

The development of malarious foci in consequence of the reclaiming of the soil, and the disappearance of the disease after its complete cultivation, are facts that have been observed on a grand scale in the most diverse parts of North America. "It is a well-known fact," says Rush (p. 97), "that intermittent and bilious fevers have increased in Pennsylvania in proportion as the country has been cleared of its wood, in many parts of the State. It is equally certain that these fevers have lessened or disappeared in proportion as the country has been cultivated." Similar observations have been made in that State more recently, as in 1849 in Bradford County, where there was an increase of malarial fever in consequence of the reclaiming of a large tract of land.[1] Statements to the same effect have been published also by Collins, Gibbs, and others for the Gulf States; by Williamson for North Carolina; by Somervail for Essex County, South Carolina, and by others. The most recent experiences, and those on the largest scale, come, as might be expected, from the Western States; they include those of Gairdner for Astoria, and of Keenay, who gives an account of the appearance of malarial fever under the above-mentioned conditions at the several military posts in Iowa,[2] and adds: "The fact not only holds good here, but has generally been so at all the various stations at which I have been, particularly at those posts where the cultivation of the soil has been one of the duties of the command;" of Logan for California, and of Stratton for Canada, where, as he states in his report, the diffusion of malarial fever has followed the progress of immigration and reclaiming of the soil from east to west, while the disease has diminished also in proportion as the country has been brought more completely under cultivation. As to the influence of the breaking up of virgin soil, there are numerous observations from Brazil (*Lallemant*, *Aschenfeldt*), from Algiers since the occupation of the country by the French (*Beaumez*, *Jacquot* (I), p. 610), from Egypt, Asia Minor, and Syria (*Pruner*, pp. 356-359), from Java

[1] Account in the 'Transact. of the Pennsylvania State Med. Soc.,' 1859.

[2] 'U. S. Army Reports,' 1856, p. 53.

(*Swaving*), and from the Banat (*Weinberger*). The remarkable increase which has occurred in the pernicious remittent fevers (*febris remittens hæmorrhagica*) within the last ten or twenty years in the Southern States of the Union is referred to by Norcom as follows: "Before the war, the Southern States were in a high state of cultivation and the lands thoroughly drained; hence the malignant forms of malarial disease as a general rule were not known, except in very low, badly-drained, swamp lands. Within the past eight years [written in 1874], owing to so much land lying waste, defective drainage, and the general unsanitary condition of the country, the malarial poison has acted with intense virulence, and caused the disease we are now considering." The same opinion is expressed by Green, by observers in Georgia, and by others.

Another interesting example of the appearance of malarial disease in consequence of deterioration of the soil in localities hitherto exempt from them, is furnished by the East African islands of Mauritius and Réunion. The fever has been observed in them since 1866, but all the authorities agree in stating that the two colonies had enjoyed a remarkable immunity from malaria up to that year. The coast and the level country of the Mauritius, which formerly afforded a rich soil for the growing of coffee, indigo, and cotton, had been gradually allowed to go out of cultivation and had become entirely barren; it thus became necessary to go higher for fruitful tracts of land to bring under cultivation, and the result has been the laying out of the sugar plantations, which have proved highly remunerative. But the deforestation rendered necessary by this new industry has led to a material change in the hydrology of the country; the mountain torrents that used to carry a great volume of water down to the coast, have either disappeared altogether or they have become so small that they scarcely reach the shore in their course, often sinking into the ground or forming small standing marshes. Then, in 1865, there came excessively heavy rains, which contributed still more to the formation of marshes; and thus there developed in 1866, under the influence of very high temperature, an epidemic of malaria which quickly spread over the whole island, and appears to have left the disease

endemic behind it. All this applies equally, as Lacaze states, to Réunion, which has always emulated the sister island in its plantations, industries, and trade.[1]

There is a scarcely less voluminous body of experience going to prove the influence upon outbreaks, or exacerbations of the disease, which has been exerted by excavations involving the disturbance of the soil to a considerable depth, such as trenches, canals, dykes, railroads, and highways, particularly when such works have been carried out on a malarious soil.[2] Improvement in the sanitary conditions of a locality by regular cultivation of the soil (whereby there are not only large quantities of water withdrawn from it, but perhaps also the processes of decomposition taking place in it are modified) has been demonstrated in various parts of the globe by the results that have followed the planting (undertaken with that object) of a highly absorbent vegetation. The first attempt of the kind, so far as I know, was the planting of the sunflower (*Helianthus annuus*) in the malarious neighbourhood of Washington; according to the account of Maury the effect has been very advantageous, and the same good result has been attained, as Martin[3] states, in some parts of the Netherlands. Experiments on a still larger scale, and attended by the same success, have been made with plantations of *Eucalyptus globulus*. The English were the first to plant that tree, at the Cape, for the purpose of drying the soil; the practice was afterwards carried out, as a sanitary measure, in several of the most malarious parts of Algiers, and, as Gimbert assures us, with equally brilliant success.[4] Summing up all these expe-

[1] See also the accounts by Barat, Nicolas, Barraut, Mereurin, Tessier, Bassignot, and Lacaze.

[2] Very interesting observations have lately (1868-70) been made on this point, according to the statement of Fokker, among the workmen employed in making the canals in Walcheren.

[3] 'Revue de thérapeutique,' 1867, Nov., p. 15.

[4] The 'Gazette hebdomad. de Méd.,' which publishes the second report of Gimbert, adds the following editorial note (1875, p. 341):—"We have ourselves had occasion to verify these properties. At the approach to the bridge of Var, on the railway, there is a guard-house built on alluvial deposits, which has been infested by intermittent marsh fever. Every now and then it became necessary to change the occupants of it. Struck by these results, M. Villard got the idea of surrounding the house with eucalyptus. Since the first year after that was done the fever has completely disappeared."

riences, Day remarks that the prevalence of malarial disease "is lessened by cultivation, increased by depopulation," and Aschenfeldt says: "Nothing but the greatest wildness or perfect cultivation protects a region from malarial fever."

Finally, I must here direct attention to the singular fact, often observed, that *changes in the soil brought about by volcanic disturbances* have repeatedly led to an outbreak of malarial disease or to a considerable increase of it. The first observations on this subject come from Italy—from Rome in 1703 (*Baglivi*, pp. 51, 388, 566), from Reggio in 1783 (*Mammi*), and from Palermo in 1828 (*Merletta*). In Peru, also, according to Smith (II) and Tschudi (pp. 440, 469) a remarkable increase of the disease has been noted after earthquakes. In recent times there has been a striking illustration of the same fact in Amboina [East Indies]; Epp, Heymann, v. Hattem and Popp, agree in stating that the island up to 1835 was subject only to a moderate amount of malarial fever of the simpler kinds, but since the earthquakes that took place in that year, the disease has undergone a steady increase, both in its area and its intensity.

§ 74. Exceptions to the Rule that Marshy Soils are Malarious.

There are few points in the etiology of disease about which so complete an agreement exists among observers, as the significance to be ascribed to the agency of the above-mentioned factors in the production of malarial diseases; the state of the case appears in fact to be so clear that it must impress the observer almost without being formally stated. The close association of malaria with a particular kind of soil, highly saturated and rich in organic matters, especially vegetable matters, and the fact that the disease breaks out whenever that kind of soil is subjected to a high temperature, suggests the conclusion that the development of the morbid poison goes hand in hand with the processes of decomposition set up in organic matters under those circumstances. This opinion will find all the readier acceptance the more

one attends to the fact that the malady prevails most on the particular soil that has all these characters most pronounced, viz. *marshy ground*, and in those tropical and subtropical regions where the processes of decomposition are most active under the influence of very great heat. It will gain in probability, also, the more one is led to admit the effect on the amount of sickness produced by those changes in the condition of the soil and climate of which we have been speaking (§ 73). However much there may be in this view that is undoubtedly correct, it has in the end led to a one-sided; theory and the theory having degenerated into a dogma, the effect has been rather to obscure than to elucidate. The facts that I am about to state will, in my judgment, serve to prove, on the one hand, that all those conditions in the soil and in the atmosphere which are required to account for malaria according to the *swamp theory*—if I may be allowed the expression—do by no means suffice of themselves to bring about the disease; while, on the other hand, the facts in the sequel serve to prove that the disease occurs both endemically and epidemically very frequently under circumstances where the effect of a saturated alluvial soil exposed to a high temperature is altogether excluded, or where the states of the soil, and the meteorological states, correspond so little to those demanded by the theory, or differ so little from the telluric and climatic conditions at innumerable other and non-malarious places, that it is impossible for us to find the cause of endemic or epidemic malaria in them alone.

Instances of non-malarious marsh.—One of the most interesting, although hitherto least noticed, phenomena in the history of the malarial diseases is presented to us in the *exemption* enjoyed by many large tracts of country, especially in the Southern Hemisphere, whose circumstances of soil and climate would lead one, on the analogy of other regions much subject to the malady, to expect its endemic occurrence. I shall limit myself to a few of the most striking facts relating hereto.

In the first edition of this work (I. p. 56) I had already called attention, although with some reserve, to the fact, vouched for by Wilson and Brunel, that the pampas of the River Plate States were quite free

from this disease, notwithstanding their resemblance in geological features to the prairies of North America and the savannas of Brazil, as well as their situation in comparatively low latitudes—notwithstanding, in short, that they afforded all the conditions necessary for the prevalence of malaria. I am now able to prove this assertion by the most certain evidence, so that there cannot well be any doubt about the fact itself. Mantegazza (I, p. 100) says, with reference to the banks of the Rio de la Plata: "Paludal fevers are nowhere known;" and he adds (p. 286), concerning the sanitary conditions in Paraguay: "Intermittent fevers have not that gravity that one might expect in such a latitude and amidst so much moisture." On this point Dupont is explicit (p. 13): "Intermittent fevers are entirely unknown along the littoral (of the La Plata); it is difficult to say to what cause their absence is to be attributed, but all physicians are agreed that they are absent. . . . . The country presents, at any rate, all the geological conditions suitable for the development of malarial fevers—undulations of the surface hardly perceptible, periodical inundations over vast tracts of land, marshes and lagoons of great extent along the banks of the rivers, great elevation of the temperature in summer." To the same purport is the statement of Bouffier: "The numerous islands of the Parana are covered with innumerable marshes, which fill and empty with the rising and falling of the river. Those marshes contain an enormous quantity of *débris*, both vegetable and animal; the bottom is generally muddy. As soon as the level of the river falls, an immense extent of marshy ground becomes exposed. For all that, I have not observed a single case of intermittent fever, and, from the information that I have been able to gather, it appears that this affection is rare among the indigenous inhabitants." Humboldt had already pointed out that the marshy banks of the Amazon in the upper part of its course are almost free from malaria, in contrast to the banks of the Orinoco and Magdalena; and that statement has been confirmed by Bates, one of the most recent and most trustworthy travellers in those parts, as well as by Galt (I). On the Peruvian pampas, also, in the upper valley of the Sacramento, malarial fever appears, from the experience of Galt (II) to be very rare. Of the *montana* region of that country Tschudi (p. 440) remarks: "We again come upon valleys with a rich vegetation, with a muggy and hot atmosphere, and covered with marshes, in which the disease is quite unknown." If we turn, now, in this survey to the eastern part of the Southern Hemisphere, we find in Australia and Polynesia a region almost absolutely free from malaria, notwithstanding that the climatic and telluric conditions, which have been frequently mentioned as favorable to the production of the morbid poison, are present there to the fullest extent. As to the complete exemption from malaria enjoyed by Van Diemen's Land and New Zealand, the opinions of Dempster (I, p. 355) and Scott agree with those of Johnson and Thomson. It is more especially pointed out by the last-mentioned writer that Europeans who have lived for years in New Zealand on the alluvial banks of the Waipa and Waikato have

remained quite free from fever, and that others who have come to New Zealand in ill health from malarious countries in the tropics have completely recovered. I have already given (p. 209) more particular details of the proportion of sickness among the English troops in that colony, and of the non-malarious condition of many of the groups of islands in Polynesia. The immunity from malaria which New Caledonia enjoys has excited the greatest astonishment among the French physicians; notwithstanding an alluvial soil abundantly saturated and covered with luxuriant vegetation, says De Rochas (p. 15), and in spite of an almost tropical climate—the mean temperature of the year amounting to 22°—23° C. (71°—73° Fahr.), and the mean summer temperature to about 26° C. (79° Fahr.)—malarial fever is almost unknown there; and, in fact, he saw during three years only a single case (of intermittent facial neuralgia) among a body of troops averaging from 90 to 100 strong, who employed themselves in road-making, trenching the ground with a view to its cultivation, hunting in the marshes, &c., without taking any precautions. Charlopin (p. 16) says that "the grand fact, which at once strikes the physician, in the pathology of New Caledonia, is the absence of intermittent fever, notwithstanding that all the conditions favorable to the existence of that malady are brought together at various parts of the island, and especially at the points most inhabited." To the same effect Bourgarel (p. 344) states: "I do not know that there has been a single case of intermittent fever, in spite of the vast marshes that are met with at the mouths of the numerous streams which water the island." Nor are striking examples of the same kind wanting in the tropical regions of the Northern Hemisphere. Thus Taulier remarks that in Manilla malarial fever has a very mild type, although the town is built upon a damp soil and surrounded by marshes and rice-fields; and Macculloch, speaking of the exemption of Singapore from malaria, says: "There is a mystery for which I can conjecture no solution, while every imaginable circumstance is present to render the land in question one of the most pestiferous spots under the sun; it is a collection of jungles and woods, marshes and rivers, and sea swamps, and it is a flat land under a tropical sun, and it is the land of monsoons, and yet it is a land where fevers are unknown;" and this assertion is confirmed in the details given by McLeod as well as in the official reports.[1] In the same way as the French physicians speak of New Caledonia, the English observers express their astonishment at the complete absence of malarial fever in the Bermudas; in the first official report, relating to the period from 1817 to 1836, we read:[2] "It is especially worthy of remark that, notwithstanding the numerous marshy situations in different parts of the island, fevers of the intermittent type are almost altogether unknown." During the twenty years referred to, there were only 27 cases in a total of 15,356 men; and, according to the report for the twenty years following,[3] there were 25 cases among 11,224 men, of

[1] 'Madras Quart. Journ. of Med.,' 1839, i, p. 64.

[2] 'British Army Reports,' 1839, 6 *b*.

[3] Ib., 1853, p. 176.

which about one half occurred in 1840, and these had been imported from the West Indies. The most recent account by Tucker entirely confirms this: "As a further proof of the marshes being innocuous, marsh or intermittent fever is unknown on the islands, which would not be the case otherwise. And, moreover, persons who have contracted fever and ague abroad often resort to this climate as a restorative." Undoubted evidence that the same circumstances occur also in higher latitudes is furnished from numerous marshy regions of North America, from Ireland,[1] which is quite free from fever in spite of its bogs, and from many parts of Sweden.[2]

§ 75. Malaria in Dry Places, Anomalous Exacerbations, Epidemics and Pandemics.

In order to estimate the importance that we should assign to wet soil, organic detritus, and such like etiological factors in the production of malaria, it is hardly less necessary to keep in view the endemic occurrence of the disease, and even of severe forms of it, in regions whose soil, in so far as relates to degree of saturation, hygroscopic properties, and richness or poverty of organic matters, does not differ essentially from the soil of many other places, sometimes in the same neighbourhood, which are either altogether free from malaria or only slightly affected by it. Classical examples of this are afforded by the hill-fever on the tableland of the Deccan, by the prevalence of severe malarial fever at a few mountainous points of Peru, by the so-called mountain fever on the slopes of the Rocky Mountains in North America, and by the endemic malaria on the west coast of Italy, especially in the Tuscan Maremma and the Roman Campagna.

"There are two errors," says Colin (p. 34), "which usually enter into the opinion that one forms of the Roman Campagna; some regard it as barren and unfruitful, others think of it as covered with stagnant water or marshes. To be disabused of the notion that it is barren, we have only to appeal to the reminiscences of those who have travelled through that country either in spring or in autumn. . . . Again, in numerous journeys made through the Agro Romano, we have become

[1] Oldham remarks that this exemption of Ireland from malarial fever in spite of the extensive tracts of marshy land "has long been a puzzle to writers on paludal poison."

[2] Bergman, p. 250. As examples he gives the country around the lakes in Ostergotland, Upland, Södermanland and Nerike.

convinced, although to our great surprise, not only that there are no marshes, but also that the soil is of extreme dryness." And there can be equally little talk of marshy soil in the Tuscan Maremma. The regions of the so-called "hill-fever" of the Deccan, the tablelands of Malwa, Chota Nagpur, and Mysore are in like manner described by observers[1] not only as free from marsh but as comparatively dry. The soil belongs mostly to the trap formation, deposited in the form of broken rock upon a granite bottom, and it contains, besides various ingredients such as quartz and felspar, a preponderant amount of ferruginous chalk. In Chota Nagpur, the soil is so porous that it becomes absolutely dry a few hours after the heaviest rain; and many of the worst fever-spots of Mysore are situated on so steep hill-sides that the water which has fallen runs off with great rapidity in cascades, the soil requiring to be irrigated artificially, and being extremely difficult to irrigate. It is obvious that no rich vegetation is to be met with in those regions. These conditions stand out still more prominently where malaria is prevalent on the bald, arid, and sterile tableland of New Castile, one of the most rainless steppes in Europe, whose scanty cultivation is kept up mostly by artificial irrigation; and, again, upon the tableland of Iran, which lies always under a cloudless sky and bright sun, and has no water from natural sources; and, further, on the plain of the Araxes, and elsewhere.

Another very noteworthy phenomenon in the history of malarial diseases, arising in this connexion, comes out in the above-mentioned fluctuations in the amount, and change in the character of the disease, in districts where malaria is endemic; and in the establishment of new malarial foci, which we can explain neither by changes in the soil nor by definite conditions of weather.

The sudden appearance of pernicious malarial fever in many parts of Chili subsequent to 1851 is an interesting example of this (see p. 222). In speaking of the endemic malarial fever at Opelousas, Cooke says (II): "The country has often enjoyed most excellent health, in spite of great and continued heats, excessive rains, and east winds, while in other years, under the same circumstances, it has been severely visited by the disease. On the other hand, there have been years in which one seemed justified in counting on a good state of health, from the mild temperature, the steady weather, scanty rainfall, and the like, but in which the disease has been very widely spread. *No one in this country is in a position, from his observations and experience, to name the circumstances which exert an unfailing influence upon the production or even the exacerbation of malarial disease.*" Precisely to the same effect is the opinion of Boling with reference to the periodical prevalence and subsidence of the disease in Alabama, and of many observers in Pennsyl-

[1] See the accounts by Ranken, Goodeve, Dunbar, and, in particular, Heyne.

vania, New York, Indiana, New Jersey, and other parts of North America. Friedlieb[1] infers, from observations made during a long series of years in Dittmarschen, that "ague may originate and become epidemic without the influence of marshy exhalations, and in all kinds of weather, and may fail to break out in marshy districts at times when the swamp atmosphere is developed to the highest degree." From similar experiences collected in Rochefort, Lucadou concludes (p. 7): "It is impossible to regard the exhalations of the marshes and the various constitutions of the atmosphere as the *sole* causes of the autumn sickness." Cameron says of the malarial fever of Ceylon (p. 72): "Certain years prove much more sickly than others, without any very evident cause."

But all attempt at explanation, in the above sense, is baffled by the outbreak and epidemic prevalence of the disease in those regions which, spared as they are from endemic malarial diseases, offer nothing in the conditions of their soil that seems able to further the production of malaria; and we are baffled still more by those pandemic outbreaks of malaria which attain a wide diffusion over great tracts of country and whole regions of the globe, and run their course, not within one season nor even within one year, but often last for several years, and then remain absent for years or tens of years.

I shall select from the overwhelming mass of facts relating to this point, two observations only, belonging to one of the latest pandemics (1858 to 1860). In his account of the circumstances of the disease in Fürth during the years 1859-60, Frohmüller[2] remarks: "Our special attention was attracted this time to ague, which broke out to an extent hitherto unknown in Fürth. . . . . Moreover, the general situation of Fürth is such that epidemics of ague had not previously been able to reach it; the elevated position of the town, between two river valleys, allows the water to run off quickly, and the Keuper bed on which the town stands, favours rapid absorption. Neither marshes nor stagnant waters lie around the town. . . . The damp character of the summer might well have acted as an encouragement, but we have often had damp summers without having had ague. A *special* factor must therefore be assumed, which it is impossible for the present to specify." Of the same pandemic, Camerer states, with reference to the disease in Stuttgart and other parts of Würtemberg that are absolutely exempt from endemic malaria:[3] "Inasmuch as ague began to be more frequent even in the hot and dry summer of 1859, it cannot be laid solely to the

[1] 'Hamb. Mag. für die ges. Heilkde.,' 1830, xix, p. 209.

[2] 'Bayr. arztl.-Intellgzbl.,' 1861, p. 45.

[3] 'Württemb. med. Correspondenzbl.,' 1861, p. 92.

account of the cold and wet of last year (1860), although the weather of that year may certainly have contributed its own share to the diffusion of the intermittent fever. On the whole, there must have been other and unknown factors contributing to bring about this form of disease."

§ 76. Ship Malaria.

Joining on to this group of facts, there is finally *the epidemic occurrence of malarial fever among the crews of ships on the high seas,* which cannot be referred to a previous infection of individuals on shore, but where we have to deal with true "ship-malaria" (Fonssagrives).[1] There have been doubtless many observations counted among these by mistake, in which the reception of the morbid poison by the patient had taken place ashore, the incubation having lasted for several weeks, as experience has not rarely shown that it may last. In going through the literature before me, I have therefore eliminated carefully all those records in which it was certain or at least probable that the disease had been acquired on land, or in which there was any sort of doubt as to the origin of the disease; and I adduce from what remain the following observations on "ship-malaria" which seem to me to be especially convincing.

On board a ship of war on a voyage from France to the Cape, an epidemic of malignant malarial fever broke out, according to the account of Laure (II, p. 12), just after the vessel crossed the line; the ports had not been open for fourteen days in consequence of very stormy weather, and the ship had become everywhere very wet owing to the openness of her planking. In this case a previous infection of the crew could be excluded with certainty. Under the same circumstances, according to Bonnaud, an epidemic of malarial fever broke out during a voyage from Toulon to Podhor, on board a very crowded frigate which had been lying for several years at Toulon and had had her bilge-water imperfectly pumped out; the crew, who had not come from malarious localities, continued to be affected for three weeks, when better weather set in and a sufficient ventilation of the hold and 'tween decks became possible. Marston[2] gives an account of an outbreak of malarial fever on board a ship bound for England from a Baltic port with a cargo of wet deals, the whole ship's company falling sick, from captain to cabin-boy; here also an infection of the individuals ashore

[1] 'Traité d'hyg. navale,' Paris, 1856, p. 218.

[2] 'Edin. Med. Journ.,' 1862, Feb., p. 709.

was certainly excluded. The following account by Holden,[1] of an epidemic of malaria on board a United States' ship of war, is especially interesting. After leaving the port of Norfolk (Va.), a pestilential stench from the bilge-water spread through the lower hold; no cases of sickness occurred among the crew, although some of them had to frequently enter the part of the hold containing the ship's stores, situated under the great cabin. A short time after, it became necessary to visit another store-room in the immediate neighbourhood of the bilge space, when the person who was sent found everything in it covered with mould. On the afternoon of the same day that person sickened with ague, and in the days following, more sickness occurred, but only in those who had entered the part of the hold which was still kept closed. The ship having put in to Beaufort, the bilges were cleaned out, and so long as the store-rooms were kept open, there was no fresh sickness; but when this regulation was afterwards disregarded, new cases showed themselves, but only among those—including Surgeon Holden himself—who had entered the room covered with mould. Facts quite similar to these are given by De Lajartre (p. 20), as observed by himself and Mairet, and by Siciliano. In the case reported by the latter, it was a French ship of war carrying troops and convicts from Toulon to Guiana and the West Indies. Here, again, the ports had to be closed in consequence of bad weather, so that the lower gun-deck could not be ventilated; and the impure state of the air in it was aggravated by a frightful stench spreading from the bilges, the gun-deck being all the while crowded with men. The first cases of malarial fever occurred as early as the second day out, and the epidemic did not cease until the bilges had been thoroughly cleaned. Something over sixty persons in all took ill, but they were almost exclusively those who were quartered in the after part of the lower gun-deck (officers, cadets, servants, &c.); while only isolated cases of sickness occurred among those occupying the upper gun-deck (passengers, convicts).

Impartial criticism cannot but conclude from all the facts above stated, that a saturated alluvial or marshy soil becomes, under the influence of high temperature, a very essential factor in the production of malaria. But at the same time malaria is not *absolutely* bound up with that etiological factor, as a *necessary* result of its activity. There is something more to be postulated, whether it be in or upon the ground or floating in the air, a *specific potentiality* which furnishes the real condition for the development of malaria, a potentiality that springs into being most easily and grows most luxuriantly under the influence of these etiological factors. But this potentiality may develop under other suitable circumstances, and quite independently of the former.

[1] 'Amer. Journ. of Med. Sc.,' 1866, Jan., p. 77.

§ 77. Necessary to assume a Specific Poisonous Substance. Search for Germs.

"Without the assumption of a material and specific malarial poison," says Griesinger very justly, "we shall not proceed far in explaining the endemicity of the fever." This declaration is primarily directed against the assumption of those observers who think themselves justified in *referring the origin of the sickness to weather influences alone*, to great diurnal range of temperature, especially when the days are very hot, the nights cool, and the air highly charged with moisture. If this theory, which was started by Maillot, Faure, Folchi, and others among the older observers, finds partisans at the present day in Espanet, Armand, Philippe, Burdel, Meyersohn, Köstler, Minzi,[1] Black, Ridrean,[2] Weir, Oldham,[3] Munro, Morrison, and others, the favour that it still finds is explained by the fact that the efforts of the former generation of observers, to bring the facts into harmony with the one-sided marsh-theory, were futile.

All the arguments of this school are directed against the marsh-theory; and, having represented it as an erroneous one, they not only make the mistake of ignoring what there is of right in it, but they come themselves to a conclusion which goes far beyond the doctrine of the marsh-theorists in its one-sidedness and its palpable errors. I hold the specific nature of malarial disease to be so generally admitted, and the belief in a specific cause underlying it to be so little open to challenge, that I do not think it necessary to enter further on a criticism of this catching-cold theory, if I may be allowed so to name it.

All that we *know* of the production of malaria forces us to assume that it stands in a close connexion with the processes of decomposition of organic matters, especially vegetable matters; and, inasmuch as the soil chiefly furnishes those matters and principally aids their decomposition, we

[1] 'Sofra la genese delle febri intermittenti,' Roma, 1844.

[2] 'Rec. de mém. de méd. milit.,' 1868, Oct., p. 289.

[3] 'What is Malaria? and Why is it most intense in Hot Climates?' London, 1871.

are led to assume that malaria is bound up with the soil in an essential degree, if not altogether unconditionally. In all decomposition (rot or putrefaction), so far at least as relates to extrinsic activity, two factors come into account: the products of decomposition, which are either gaseous or solid matters, and the excitants of decomposition whose organised nature cannot well be longer doubted. Corresponding to these assumptions, there have been certain theories hitherto current as to the nature of the malarial substance. One view is, that it is represented by a kind of gas or gaseous mixture; the other view runs into the zymotic theory, on the one hand, and the parasitic theory on the other.

The first-named and oldest of these, already hinted at by Varro,[1] afterwards laid down by Lancisi,[2] extended by Baumes[3], and adopted by Savi, Daniell, and Bonssingault, has found a few adherents even in the most recent times. It is based upon the detection of hydrogen compounds of sulphur and carbon in the air of the marsh, and particularly upon the fact that volcanic soil is a favourite seat of malarial foci, and that the disease occurs so frequently in the very neighbourhood of active volcanoes. This theory has lately been adopted by Schwalbe, who explains the malarial poison to be carbonic oxysulphide, according to his observations made in Central America. He assures us that carbonic oxysulphide may be detected by the smell on the Isthmus of Panama; but he adds that the experiments which he has made upon animals with that gaseous mixture have not given a positive result, and that there are perhaps still other gases or gaseous mixtures in which the cause of the disease might be found. Against this theory, there is the fact that the effects of the different gaseous combinations are well known, and that none of these effects correspond to the peculiar phenomena of malarial sickness. Further, it is certain from the

[1] 'De Architectura,' lib. i: "Spiritusque bestiarum palustrium venenatos, cum nebula mixtos, in habitatorum corpora flatus spargent, efficiunt locum pestilentem." It is certainly doubtful whether Varro here implies by the word "spiritus," the breath of poisonous animals or a development of poisonous kinds of air from the decomposition of the latter.

[2] 'De noxiis paludum effluviis,' &c., Colon,' 1718.

[3] 'Mem. sur les maladies qui résultent des émanations des eaux stagnantes et des pays marécageux,' Paris, 1789.

inquiries made in the malarious regions of the Sologne by Lafont (p. 9) and others, that the volume of these gases in the air of marshes ranges from little to nothing, and that owing to their diffusibility, they can act only in a very diluted state. Lastly, the prevalence of malarial fever is by no means proved in the neighbourhood of the localities where the aforesaid gaseous combinations are mostly detected, as in the vicinity of certain manufactories, sulphur mines, and the like. There remains only the conjecture that the malarial poison is represented by hitherto undiscovered gases or mixtures of gases, capable of producing their specific effect in the highest possible state of attenuation, an assumption for which there is at present no actual foundation.

The *zymotic theory* is based upon the doctrine of ferment-action. It coincides, therefore, in parts with the parasitic theory; but, in contrast to the latter, it leaves the question open, whether it is the excitants of decomposition as such, or whether it is the formed products of decomposition or the already defunct fermentative producers, that act as poisons, producing the special pathogenetic effect upon the organism. Of all the theories that have hitherto become current as to the nature of the malarial poison, that which asserts the *parasitic character of the disease* meets with most approval at present, inasmuch as it is supported by other analogous facts in the etiology of disease, and has at least this preference that it satisfactorily explains the peculiarities in the occurrence and course of malarial fever as an endemic and as an epidemic.

Mitchell[1] was the first to approach in a scientific spirit the question of the parasitic nature of the infective diseases, and particularly of malarial fever. Soon after the appearance of his work, Barnes[2] also declared that inquiries into the malarial fever in Fort Scott, Kansas, gave much probability to the assumption of the parasitic nature of the malarial poison; and to the same effect was the opinion of Gigot from his observations made in the department of the Indre. Lemaire[3] examined with the microscope the mist that rose and condensed over the marshes of the Sologne, and satisfied himself that it contained abundant

[1] 'On the Cryptogamous Origin of Malarious and Epidemic Fevers,' Philad., 1849.

[2] 'U. S. Army Reports,' 1859, p. 163.

[3] 'Compt. rend. de l'Acad.,' 1864, i, p. 426, ii, p. 317.

lower organisms which grew out of the cells and spores contained in the moisture, and seemed to be related to the malaria there prevalent. Then came the statement of Massy, that at a time when severe malarial fever was prevalent in Ceylon, he had found a (microscopic) fungus which floated in enormous masses in the air, was precipitated everywhere, was even found in the urine and sputa of the sick, and represented, as he believed, the true malarial poison. In like manner Baxa and Wiener thought that they were justified in assuming that, among the lower organisms met with in the marshes of Pola, one in particular, having the form of a simple cell, was the malarial fungus. Holden explained the above-mentioned outbreak of malarial fever on board a ship of war (p. 285) as follows :—The algæ belonging to the family of Thallophytes, which he had found in the infected store rooms, were harmless in themselves, but they had taken on poisonous properties from combining with the sulphuretted hydrogen set free under the circumstances named, and had so produced the disease. Just about the time of Holden's announcement, there appeared the well-known paper of Salisbury, adducing evidence that in the soil of malarious foci on the banks of the Ohio and Mississippi, there grew a species of alga (Palmella) whose spores were carried into the atmosphere by the ascending current of air; they thus came within reach of the respiratory and digestive organs of the individual, and he thought to prove by an experiment, which we shall mention afterwards, that they were the malarial poison. The announcement of Salisbury excited general attention, and if the conclusions which he drew from his studies did not escape some well-founded objections, they were at the same time abundantly confirmed. Thus Van den Corput[1] stated that, when he was a student, he had been several times ill with malarial fever while growing algæ and other marsh plants in his bedroom; and Hannon[2] declared that he had observed the same thing himself in 1843 at a time when he was engaged in the study of the fresh-water algæ. Shortly after, Balestra published the result of his inquiries into the algæ present in the Pontine Marshes; he showed that, besides numerous low organisms in the water, there was one species of alga that grew with enormous rapidity when it was exposed to air and light, and whose spores could be detected in the atmosphere over the Pontine Marshes, as well as over the Roman Campagna. He took the fever himself twice after drawing deep breaths over a vessel containing marsh water so infected; and he satisfied himself also that, on the addition of sulphate of soda, arsenious acid, or sulphate of quinine, not only did the reproduction of these algæ cease, but they and their spores underwent a change in their structure; and this led him to think that there need be no hesitation in designating these microphytes or their spores as a true cause, and perhaps the only cause, of malarial fever. Selmi[3] came to almost the same conclusion in his investigation of the mist overhanging the marshes of Mantua. In a

[1] 'Journ. de méd. de Bruxel.,' 1866, March, p. 330. [2] Ib., May, p. 497.

[3] 'Il miasma palustre,' Padova, 1871. (Also given in abstract in 'Il Morgagni,' 1873, p. 437.)

case of malarial fever observed by Schurtz[1] at Zwickau, where the disease is extremely rare, the question was to account for the illness of a person who cultivated Oscillaria in his bedroom in the course of his botanical studies; the Palmella, as Schurtz conjectures, may have some genetic relations to the Oscillaria. The observations of Salisbury were further confirmed by Bartlett,[2] who found the microphytes described by the former in enormous diffusion in the malarious localities on the Mississippi near Keokuk, where the disease was universal: "The course of this disease," said Bartlett, "seemed *pari passu* with that of the plant." Magnin, who has investigated this question in the marshes of the Dombes, is also of opinion that the cause of the disease is to be referred to a microphyte, one of the species of Oscillaria; while Lanzi,[3] basing on his observations made in the Roman Campagna and the Pontine Marshes, throws out the conjecture that the formation of the malarial poison is a matter of peculiar degeneration of the cells of algæ, which become filled with black granules, identical perhaps with the sphaerobacterium of Cohn or the bacteridium brunneum (Schröter), and constituting the true infecting matter. Klebs and Tommasi-Crudeli believe that the question of the production of malarial diseases, by the taking up of low vegetable organisms, has been finally settled by their experiments in the Roman Campagna and in the Pontine Marshes. Both in the soil and in the air of these malarious localities, they found a kind of bacillus in the form of rods and elongated oval moving spores, which, when isolated and cultivated, produced the most marked malarial sickness in the animals which received them. The fevers varied from the mildest to the most intense or so-called pernicious kind, fatal in twenty-four hours; the firm swelling of the spleen and the melanæmia which were observed at the same time, afforded further evidence of the identity of these artificially produced fevers with the malarial sickness occurring in man. The absence of this bacillus malariæ from the stagnant water of those parts, although it was unusually rich in other lower organisms, was also accounted for by the experiments; large quantities of water either prevented the development of this malarial poison altogether, or rendered the existing germs of the disease powerless. In the body of the infected animal the bacillus was most abundantly developed in the spleen and the marrow of the bones, where in some cases there were long homogeneous threads from 0·06 to 0·08 mm. in length and 0·6 micro-millimetres in thickness. Marchiafava has found the same bacillus malariæ in the bodies of several persons who died in Rome of pernicious malarial fever, and Griffini has confirmed the occurrence of them in the Lombard rice-fields.

It need hardly be said that, with all these observations, the question of parasitic malaria is not yet absolutely solved; gross errors underlie some of the observations,

[1] Archiv der Heilkunde, 1868, p. 69.

[2] Brit. Med. Journ.,' 1873, Jan., p. 54.

[3] Med. Times and Gaz.,' 1876, Dec. 2, p. 625.

particularly those of Salisbury, as Wood,[1] Harkness,[2] and Weir[3] have shown. It must rest with the future to decide upon the value of this theory.

A peculiar theory of malaria, assigning to it a telluric origin, has lately been set up by Colin. In his view, it is essentially an affair of a power issuing from the soil, a "*puissance végétative du sol*," which becomes a cause of disease when the power is not exhausted by cultivated plants. "So far," he says,[4] "from having to search in the vegetation of the marsh for the cause of the fever, I believe that it is rather in the inverse condition, in the absence of this vegetation, that one is likely to find it. In my view, indeed, the fever is caused most of all by the vegetative power of the soil whenever that power is not called into action, when it is not exhausted by plants sufficiently abundant to use it up," and in this sense he designates the disease as an "*intoxication tellurique*."

Finally, there have not been wanting attempts to refer the origin of the disease *to the toxic excretions of living organisms, plant or animal*, instead of to the products of decomposition of organic matters or to parasitic bodies. Boudin[5] has conjectured that the ethereal oil excreted by certain plants, especially *Anthoxanthum odoratum*, *Chara vulgaris*, some rhizophoræ, and others, represents the malarial poison; and this view appears, from the statement of Vaughan,[6] to have been lately received with favour in the Academy of Medicine of Cincinnati. Bouchardat[7] has set out with a similar idea, only that he substitutes the animal kingdom; he believes that the malarial poison is an excreted product of certain infusoria vegetating in marshes or in a damp soil. "This is a hypothesis," he adds, "which gives the best account of the facts observed; to speak of this substance as allied to the poisons introduced by animals (*les venins*) is only to assign to the facts their most legitimate interpretation." The innocuousness of certain marshes is explained by Bouchardat

[1] 'Amer. Journ. of Med. Sc.,' 1868, Oct., p. 333.
[2] 'Boston Med. and Surg. Journ.,' 1869, Jan., n.s., ii, p. 369.
[3] Ib., 1870, Dec., n.s., vi, p. 390.
[4] 'Traité,' p. 14.
[5] 'Traité des fièvres intermittentes,' Paris, 1841.
[6] 'Philadel. Med. and Surg. Reporter,' 1871, Dec., p. 551.
[7] 'Annuaire de thérapeutique,' 1866, p, 299.

in this wise: either that they do not contain those "infusoires toxifères" at all, or that certain vegetable matters occur in them coincidentally, which nullify the poisonous effects of the infusoria.

However far we may still be in science from getting a definite answer to the question of the nature of the malarial poison, and on however inadequate grounds all the numerous attempts designed to explain it may appear to rest, there is one idea running, like a red thread, through all those hypotheses, which seems to me to be of fundamental importance for the final solution of the problem. Whether we regard the malarious substance as a product of the decomposition of organic matters, or as parasites, or as an animal or vegetable poison, or in what way soever, we shall always ascribe *specific* properties to it, and always come therefore in the end to some *specific source whence it has arisen*. We are therefore driven to the supposition that the morbid poison develops only from, or within, certain organic matters (animal or vegetable); and so the inquiry directs itself in the first instance to the study of the lower fauna and flora both of the localities were malarial fevers are endemic, and also of those which are exempt from the disease, with a view to ascertaining, from a comparison of the results so obtained, first of all what animal or vegetable forms the production of malaria appears to be associated with. It is not the *amount* of vegetation (which has hitherto so fixed the attention of inquirers) as the *specificity* of it, which ought, in my opinion, to come mostly into consideration; it is this specificity that appears adapted likewise to explain the prevalence of the disease in some very barren regions, as well as its absence from other tracts of country with a luxuriant vegetation; and, finally, to explain the fluctuations in the amount of sickness, the appearance of it as an endemic when agriculture is neglected, and the disappearance of it when the cultivation of the soil has been changed or reduced to greater regularity. Along with the knowledge of such living species, we ought to aim at a deeper insight into the pathogenetic influence of the soil itself, inasmuch as both its physical and geological peculiarities may determine the character of the fauna and flora supported by it.

§ 78. One Malarial Poison or Several?

So long as we remain ignorant of the nature of the malarial poison, we shall be working merely on a basis of probabilities in attempting to answer the question whether there are *various morbid poisons* underlying the various forms of malarial disease, or whether there is only *one* malarial poison, whose kind of effect is various according to the quantity in which it acts, and according to the predisposition of the individual affected by it. From the standpoint of criticism, I must decide in favour of the second notion; because experience shows that the disease, both in the individual and in the epidemic, exhibits transitions from one form to the other, that the intensity of the disease at one and the same place depends upon vicissitudes of external conditions, especially heat and moisture, and that the form of sickness stands in a definite relation to the power of resistance in the organism that has been invaded by the morbific cause. These are the considerations that justify us in bringing together all the forms of disease here spoken of into the single category of "*malarial disease.*"

§ 79. Alleged Diffusion by the Wind—Absence of Contagious Power.

There can be no doubt that the malaria generated in the soil may exert its influence at a distance from its centre of origin, and that this *diffusion of the morbid poison is effected by the wind.* Lancisi was the first to throw out the idea that the wind may be regarded as a carrier of the malarial poison;[1] and even if the assumption on which he built his opinion was an erroneous one,[2] there have been since then very many and

[1] P. 93.

[2] Lancisi, as is well known, believed that the endemic malaria of the Roman Campagna owed its origin to the emanations of the Pontine Marshes finding free access to the Campagna in consequence of the cutting down of the sacred groves during the pontificate of Gregory XIII. He had forgotten, however, that between those marshes and the Campagna there are the Alban Hills, which would afford a much more certain protection than the woods, supposing always

unambiguous observations to prove that the appearance and prevalence of the disease is often subject to the influence of winds blowing over marshes and other sources of malaria, and carrying the poison to places otherwise exempt from the disease; it is also proved that ascending currents of air may carry it to certain altitudes, while, on the other hand, barriers of various kinds, such as hills and elevations of the ground, belts of trees, walls, and the like, lying in the track of the winds charged with malaria, may afford a protection from the poison to the localities situated behind them.

Facts of this class are reported from almost all malarious regions. Thus Le Gendre (p. 26) observes that the hill country in Medoc is visited by malarial fever only when it is exposed to the winds passing over the adjoining marshy plain, but never when the opposite wind is blowing; and similar observations are published by Cornay for Rochefort and by Croigneau for Rochelle. Mondineau states (p. 15): "It is quite certain that in the districts of the Canton of Houeilles, intermittent fevers have become much more rare, and everywhere less severe since the immense forests of shore pines have grown up to form a natural barrier to the propagation of the miasm." Jilek (p. 59) shows that, in Pola, those parts of the town suffer chiefly from malaria which are most exposed to the winds blowing from the neighbouring marshes. Moore points out (p. 289) that the island-like plain of Cutch, situated close to the salt-marshes of Rann, must needs be infested by the severest malarial fevers were it not that for ten months of the year the wind blows from the island to the marshes. Wilcocks found, in the severe epidemic of malarial fever that prevailed in Philadelphia in 1846, that the streets and rows of houses in the quarter of the town most affected, were nearly always those exposed to the wind from the south. Coons gives the following interesting case from the epidemic of 1826 in Alabama: Near Moulton, and half a mile from a lake, there is a large farm whose inhabitants enjoyed excellent health up to 1826; in the summer of that year, a dense wood which separated the farm from the lake was cut down, so that the plantation was exposed to the winds blowing over the marshy lake, and a year after, so intense an epidemic of malarial disease broke out that only three or four out of the 150 persons on the plantation escaped the sickness. Observations to the same effect are given by Wooten for Lowndesborough:[1] here, also, it was an affair of a plantation separated by a thick belt of wood from the swampy shores of a creek a quarter of a mile away; the wood was cut down in the winter of 1842-3, and in the following summer the negro hands on the plantation, who had hitherto escaped the malarial sick-

that the exhalations from the marshes can in fact make themselves felt at so great a distance.

[1] Lewis, 'Med. Hist. of Alabama,' p. 17.

ness, suffered so much from it, that the proprietor was obliged to quarter them on the other side of the river, which was still sheltered by a wood; whereupon the sickness abated and the former state of health was restored. Jackson (III, p. 61) publishes the following observation made by him in 1778 in Jamaica: near King's Bridge, some two hundred paces from a swamp, a camp was pitched on a dry and tolerably elevated piece of ground which was exposed to the winds blowing across the swamp; it was especially the more elevated part on the right that was struck by the wind, and malarial fever very soon broke out there, while the lower part on the left remained almost exempt from it. The soldiers whose tents were pitched on a height in front of the camp suffered most, being most exposed to the effects of the wind, and none of them escaped the epidemic.

It would be hardly possible to find a numerical expression for the distance to which malaria may be carried by the wind; it is most probable that it is limited to a very small range relatively. At least all the most reliable observations made on land bear this out, while experiences as to the diffusion of the disease from the shore to vessels anchored in the immediate neighbourhood is still more conclusive. All those experiences go to show that the crews of ships lying so close to the shore or the coast as to be necessarily affected by the land wind, are almost always exempted from the disease so long as they do not set foot on the infected shore; and this applies even to the most intense malarious spots.

Lind long ago remarked: "Noxii vapores, qui paludibus emanent, haud longe patent; nam persæpe naves a littore haud multum remotas a labe prorsus immunes vidimus." The fact has been subsequently confirmed by Badenoch and Allan for the Comoro Islands and the coast of Madagascar, and by Ritchie and Griffon for the Congo Coast, to this extent at least that only the very closest proximity to the shore has any influence on the crew, and then only in a very limited degree. "When the ships watered at Rock Fort," says Blane,[1] "they found that if they anchored close to the shore, so as to smell the land air, the health of the men was affected; but upon removing two cables' length, no inconvenience was perceived." Rattray makes the same remark for ships lying in the harbour of Hong Kong: "While the fever . . . . was fatally prevalent on shore, the ships

[1] 'Observations on the Diseases incident to Seamen,' Lond., 1799, p. 221.

in harbour, even when lying at very short distances from the shore, are usually or often exempt from its ravages."

Least of all is one justified in attributing to a diffusion of malaria by atmospheric currents, the great malarial epidemics and pandemics, whose origin for the present admits of no adequate explanation founded on facts; in not a single case has the disease on a large scale been shown by observations to have spread according to the direction of the wind. In like manner, all those observations which have been adduced to prove the diffusion of malaria by means of drinking-water, do not, in my view, bear the construction that the writers put upon them.

Question of drinking water.—Thus Jussieu,[1] in his account of the epidemic of malarial fever in Paris in 1731, makes out the cause to have been the use of Seine water contaminated by rotting water-plants and confervæ. Meynne (p. 364) communicates some observations from Belgium on an outbreak of malarial fever in consequence of partaking of marsh water. Similar statements are made by Perier (p. 10) for Algiers, and by Lord (p. 461) for the delta of the Indus, where the natives are so convinced of the infective properties of well water that they say no stranger can drink it for two weeks together without taking malarial fever, and the author adds that he has had occasion to assure himself of this fact during a halt of the English troops for only a few days in Bahawal Khan. Reumert[2] narrates in his account of the medical topography of Fridericia, that there occurred in 1855, in a quarter of the town much affected by malarial fever, a series of cases limited to such persons as had taken their drinking water from a particular spring. Parkes[3] mentions that he visited the marshy plain of Troy during the Crimean War, and there discovered that those of the inhabitants who drink marsh water suffer from malarial fever all the year, while others who use pure spring water suffer only in summer and autumn. Boudin[4] gives the following: On board a French ship of war bound from Bona (Algiers) to Marseilles, a malignant epidemic of malarial fever broke out at sea, thirteen men dying out of a crew of 229, while ninety-eight were more or less severely ill, and had to be sent into hospital at Marseilles; it came out, on inquiry, that the vessel had shipped at Bona several casks of marshy water, which had given occasion to lively dissatisfaction among the crew on account of its disagreeable smell and taste, and that not a single case of sickness had occurred among those of the crew who had drunk pure water.

[1] Mém. de l'Acad. des Sc. Ann.,' 1733, p. 351.
[2] 'Danske Sundhetscoll. Aarsberetning for 1855,' p. 67.
[3] 'Manual of Practical Hygiene,' London, 1869, p. 71.
[4] 'Géogr. et statist. méd.,' Paris, 1857, i, p. 142.

To all these statements, and many more like them, there is, in my opinion, no definite value to be attached for answering the question at issue; and that is also the opinion which Colin has expressed in the most decided way on the strength of his experiences in Algiers and at Rome. Much of this so-called experience rests plainly upon mistakes, or at least on erroneous suppositions; in every case, it is a question of individuals sickening who had been otherwise exposed to malarious influences; and even if one were to admit that the partaking of putrid or marshy water may have an influence on the production of malaria, the question is not one of *specific* cause, but of such an injurious influence as would increase the predisposition of the individual to malarial sickness, or to the occurrence of relapses, by lowering his power of resistance.

The notion that a *contagium develops in the course of the malarial process*, or, in other words, that the morbid poison reproduces itself within the body of the malarial patient, gets eliminated therefrom, and conveyed to other individuals, whereby it brings about the diffusion of the disease—this notion must be dismissed in the most decided way, according to the opinion of all observers and the experiences of all the great epidemics.[1] But it is otherwise with the question whether there is any conveyance of the malarial poison, engendered in or upon the soil, by means of the soil itself, or of other objects to which this poison may cling. It is somewhat remarkable that no attention has been given to that particular statements of Salisbury's on the parasitic character of the malarial poison, which, in my opinion, appears to be the most important of them all, assuming always the trustworthiness of the facts. I refer to the observations which seem to furnish proof that *the malarial poison may be conveyed from place to place.* In order to prove the fever-producing property of the palmella, he devised the following experiment:

He filled several boxes with earth taken from an extremely malarious soil and abundantly penetrated and covered with algæ; he then took

[1] The observations adduced by Thomas ('Archiv der Heilkd.,' 1866, p. 307), to prove the contagiousness of malarial fevers (probable conveyance of the disease from infants to nurses and *vice versâ*) cannot well be regarded as convincing.

them to a house about 300′ above the level of the river, in a perfectly dry situation, distant some five miles from the nearest malarious locality, and where there had never been a case of malarial fever. They were placed on the window-sill of a room on the second floor, used as a bedroom by two young men, and care was taken that the window should not be closed during the night. On the sixth day after the boxes were deposited, both the inmates of this room complained of being unwell; on the twelfth day, one of them had the first decided attack of ague, and the other on the fourteenth day; the fever took a tertian type, and soon disappeared under the use of quinine; all the while four members of the family who slept in the lower rooms of the house escaped the disease. A second experiment resulted the same way; it took place at a building adjoining the house already mentioned, in a room occupied by a man and two boys; on the tenth day one of the boys took intermittent fever, and the other boy on the thirteenth day, but the man continued well.

It must remain an open question whether palmella spores contained in these boxes and conveyed by the currents of air into the room, gave occasion to the sickness; at all events the assumption appears well founded that something was brought into the rooms in the boxes containing the earth, which had a morbific effect. In that sense also we may perhaps interpret an observation lately recorded by Sawyer.[1]

The author, who resides in a malarious part of Illinois, visited a friend at Milton, Mass., and fell ill there of intermittent fever. The lady of the house, who interested herself greatly on the patient's account, and who had never seen a case of ague before, had a slight aguish attack of fever and chill on the fifth day, with gastric disturbance; but she set aside the idea that she could possibly be ill of ague, as the disease was quite unknown in Milton, or occurred only now and then in imported cases. However, she had a more severe attack the day after, and on the ninth day the first pronounced paroxysm of ague occurred, and, with that, all doubt as to the nature of the disease vanished.

The possibility of the patient having brought the malarial poison with him from home in his clothes or other effects, and so given rise to the lady's illness, cannot on the face of it be contested; and thus the question arises whether the malarial poison is not transportable, and whether we may not perhaps interpret the development of malarial epidemics in places otherwise exempt from the disease, as well as the formation of new endemic foci, in the sense that the morbid poison, somehow conveyed, has landed on a soil that renders

[1] 'Boston Med. and Surg. Journ.,' 1867, Dec., p. 538.

its reproduction possible, either for a time or permanently. We may find a corroboration of this in the outbreak and spread of the disease in Sweden (Bergman), Finland (Hjelt), and other countries; and it may be that, from this point of view, Lacaze is right when he says of the malarial epidemic that broke out in Réunion in 1868: "The fever had existed barely three years in Mauritius, when the first cases were observed in Réunion. There is a probability, amounting almost to a certainty, that an importation had taken place thither from the Mauritius."

The last question belonging to the history of the malarial diseases relates to the alleged antagonism in space or area between those diseases on the one hand, and typhoid fever and consumption on the other. This question will be duly noticed in the accounts of the latter diseases.

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CHAPTER VIII.

YELLOW FEVER.

§ 80. Peculiar Geographical Distribution. Chronology of the Epidemics.

Measured by the range of its distribution over the earth's surface, yellow fever takes one of the lowest places among acute infective diseases. If we disregard its isolated appearances at several points on the western shores of Europe, and its quite recent establishment as a prevalent form of sickness on the Atlantic and Pacific coasts of South America, there remain only two among the greater regions of the globe to form the seat of yellow fever: on the one hand, the shores of the Gulf of Mexico, including the West Indies, and part of the Atlantic coast of the United States, and, on the other hand, a part of the West Coast of Africa.

The earliest history of yellow fever in the former of these two regions is enveloped in an obscurity which we cannot now enlighten. In particular, there is no way of getting an answer to the question whether the disease was prevalent there, and, if so, to what extent, before the arrival of European colonists; or to the question, when and under what circumstances it first appeared among the new comers. It is not only the defectiveness of the records from these earliest times of our intercouse with the Western Hemisphere, that renders all historical research on the subject illusory; a still more serious impediment, and one that has continued in force down to the most recent times, is the frequent confounding of yellow fever with bilious remittent malarial fevers, especially with febris remittens hæmorrhagica. All the observations dating from

the sixteenth century, by Herréra,[1] Dutertre,[2] Rochefort,[3] and others, on the destructive pestilences which had raged among the natives of the West Indies before the arrival of Europeans, as well as the accounts of Herréra,[4] Oviedo,[5] Lopez de Gomara,[6] and others, as to the disastrous sickness among the first settlers in Domingo in 1494 and 1504, in Porto Rico in 1508 and 1513, and on the Gulf of Darien in 1509 and 1514, afford scarcely any safe indication of the nature of the disease, for the reason that they are derived from non-medical observers; while it is at the same time certain that the Europeans (Spaniards, Frenchmen, and Englishmen) suffered, soon after settling in North and South America, from the severest forms of malarial fever, to such an extent that numerous settlements were depopulated in a few years, and whole armies perished. Still less credence is due to the opinion adopted by Webster[7] that the severe pestilences mentioned by Hutchinson[8] and Gookin[9] as having occurred during the sixteenth century among the natives on the East Coast of North America, are to be taken as yellow fever; for it is expressly stated that they prevailed in the winter time, during severe cold, exclusively in the wigwams of the Indians, the European settlers escaping them. Neither is there any reason for identifying with yellow fever the Mexican pestilence mentioned by Humboldt[10] under the colloquial name of "Matlazahuatl;" for that also was prevalent, in the epidemics that are known (1545, 1576, 1736-37, 1761-62), almost exclusively among the natives of the country, and it affected only the interior and the table-land of Mexico, sparing the coast regions.

The first reliable accounts of yellow fever date from the middle of the seventeenth century; they tell of the importa-

[1] 'Historia general de los Echos de los Castellanos in las Islas y Terra firma del Mare Oceano,' Madrid, 1601.

[2] 'Hist. génér. des Antilles Franç.,' Paris, 1667.

[3] 'Hist. naturelle et morale des Iles Antilles,' Lyons, 1667, ii, p. 475.

[4] L. c., bk. i, chap. 10, 12; bk. x, chap. 14.

[5] 'La Historia general des las Indias,' Madrid, 1547, bk. ii, chap. 13.

[6] 'La Historia des las Indias,' Medina, 1553, bk. i, chap. 2.

[7] 'History,' i, p. 176.

[8] 'History of Massachusetts,' i, p. 34.

[9] 'Historical Collections,' p. 8.

[10] 'Essai politique sur le Royaume de la Nouvelle Espagne,' i, p. 352.

tion of the disease from place to place, and from island to island; and they advert to the circumstance that it was especially the new arrivals that were visited by the sickness most, and that not only the settlers but also the crews of merchant ships and ships of war at anchor in the ports of the West Indies were menaced to a great extent by the disease. In the following pages I have represented in tabular form the epidemic history of yellow fever in the above-named regions (American), as far as it has become known to us; and for the better surveying of it, I have arranged the epidemics in two tables, the one giving the general chronology, and the other the localities (with dates) in the order of their latitude.

I. *Chronological survey of the epidemics of yellow fever, in the West Indies, North America, Central America, and the Mexican Gulf Coast of South America.*

1635	Guadeloupe: Dutertre i, 81.
1640	Guadeloupe: id. 99.
1647	Barbadoes: Ligon (History of Barbadoes, Lond., 1657) p. 36.
1648	Guadeloupe: Dutertre i, 423.—St. Kitts: id., ib.[1]
1652	St. Kitts: Maurile de St. Michael (Voyage des Iles en Amerique, 1652) p. 45.
1655	Jamaica: Moseley, 422.
1656	Domingo: id., ib., and Moreau (I), 60.
1665	Sta. Lucia: Dutertre iii, 86, 244.
1671	Jamaica: Trapham (State of Health of Jamaica in 1679, Lond.), p. 81.
1688	Martinique: Labat (Nouv. Voyage aux Iles de l'Amérique, Paris, 1722) i, 72,
1690	Barbadoes: Hughes (Natural History of Barbadoes, Lond., 1750) p. 37.—Domingo: Moseley, Moreau, ll. cc.—Santa Cruz: Moreau, l. c.
1693	Martinique: Labat.—Caracas: Humboldt (Voyage) ii. 400. —Boston: Webster i. 207; La Roche (II) i, 48.[2]—Philadelphia: La Roche (II) i, 49.—Charleston: id., ib.
1694	Barbadoes: Ligon.
1696	Domingo, Martinique, and other West Indian Islands; Moreau (I), p. 53.—Venezuela.

[1] See also Webster (in the 'New York Med. Repos.,' vii, p. 322), who supplies information about the epidemic from the manuscript records of the historian Hubbard.

[2] It is questionable whether this account relates really to Boston, or not more probably to a more southern port, New York or Newport.

1699	The West Indies over a wide extent: Bally (I), p. 36.—Vera Cruz (first epidemic): Heinemann (II). — Bermuda: Barrow, p. 290. — Philadelphia: Webster i, 211; La Roche (II) i, 50.—Charleston: Webster i, 212; Ramsay (I), p. 96.
1702	New York (August—Sept.): Webster i, 217; Bard.—Bay of Biloxi (first French colony on the Gulf Coast): Drake ii, 215; Lewis (II).
1703	Guadeloupe, Martinique: Labat. — Charleston: Hewatt (Account of the Rise and Progress of the Colonies of S. Carolina, &c., Lond., 1779) i, 142.
1705	Domingo, Martinique: Labat, Feuillé (Journ. d'observ. dans la Nouvelle Espagne, &c., Paris, 1725), p. 186.—Mobile: Lewis (II).
1706	Martinique: Feuillé, p. 187.
1715	Barbadoes: Chisholm ii, 177; Brown (II), p. 12.
1721—24	Barbadoes: Warren (I), Toune.
1725	Vera Cruz: Clavigero (Hist. de la Mexique) i, 117.
1726	Porto Bello: Ulloa (Voyage histor., Paris, 1752), p. 84.
1728	Charleston (in Summer): Hewatt, p. 317.
1729	Antigua: Bally (I) p. 40.—Porto Bello: Ulloa.
1731	Domingo (on board the fleet arrived from Porto Bello): Bancroft (I), p. 336.
1732	Charleston (May—Oct.): Ramsay (I), p. 36 and (II), Moultrie.
1733—39	Barbadoes: Warren (I), p. 4.
1733—37	Domingo: Desportes i, pp. 40, 66, 74, 86.
1737	Norfolk (Va.): Mitchell (I).
1739	Charleston (August and fol.): Ramsay (I), p. 36.
1740—	Cartagena, Porto Bello, Panama, Vera Cruz: De Gastelbondo (Tratado del Méthodo curativo . . . . de la Enfermedad del Vomito negro, &c., Cartagena, 1753).
1740	Domingo (June—Dec.): Desportes i, 97, 110, 114.
1741	Domingo (June—Dec.): Desportes i, 97, 110, 114.
1741—42	Jamaica (in the English fleet on its arrival from Cartagena): Hume (I), p. 230, Williams (I), p. 11.
1741	Norfolk (Va.): Mitchell (I).—Philadelphia (July and fol.), Currie (I), p. 211.
1743—46	Domingo (Summer and Autumn): Desportes i, pp. 129, 133, 136, 138, 166.
1743	New York (July—Oct.): Webster i, p. 238. — Newhaven: Munson, in 'Additional Facts,' &c., p. 54.
1745	New York: Webster i, 239, 341.—Charleston (June—Sept.): Ramsay (I), p. 39, Moultrie, p. 165.
1747	Philadelphia (June—Oct.): Currie (I), p. 212, Pemberton in 'Additional Facts,' &c., p. 6; Chew, ib., p. 11, Bond, Bartram.
1748	Charleston (August—Oct.): Moultrie, p. 171; Lining, p. 419.
1750	Martinique: Bally (I), p. 49.
1751	Curaçao (April): Winds in Lind, p. 110.
1754	Antigua (Autumn): McKittrick, p. 107.
1756	Antigua: Lind, p. 185.
1760	Surinam (first epidemic): Fermin.—Curaçao (in autumn): Rouppe, p. 303.
1762	Havana: Moreau (I), p. 73, Webster i, 251. — Philadelphia (August—Nov.): Redman in 'Additional Facts,' &c., p. 19; Willing, ibid., p. 9, Rush (I), p. 15.

1763—65 Cayenne: Bajon ii, 46; Campet, p. 73.
1765 Mobile: Romans ('Hist. of Florida,' Philad., 1776), p. 13; Drake ii, 216. — Pensacola: Lind, p. 37, Romans.
1765—66 Antigua: Lind, p. 213.
1767 Sta. Lucia: Leblond, p. 154.
1770—71 Grenada: Leblond, pp. 166–7.
1770—72 Martinique: Moreau (I), p. 82, after Rochambeau.
1779—80 Bermuda: Barrow, Smart.
1780 Jamaica, St. Lucia: Bally (I), p. 56.—Havana: Caillot.
1781—82 Havana, St. Domingo: Berthe, p. 164, after Fr. Balmis.
1783 Baltimore: Webster i, 274.
1791 Cayenne: Leblond, p. 209. — New Orleans (first epidemic): Drake ii, 201. — New York: Addoms, Carey, p. 85.
1792 Charleston (August—Sept.): Ramsay (II), Harris (III).
1793 Caracas (October): Rush (I), p. 390.—Porto Bello: Moreau (I), p. 156. — Demerara, Trinidad: Chisholm ii, 201, 228. —Tobago (July): Ibid., p. 212. —Grenada (March—Sept.): Ibid., i, 96; Stewart, Smith (II).—St. Vincent (April): Chisholm ii, 144.—Barbadoes (May): Ibid., p. 180; Romay, 168. — Martinique (October): Chisholm ii, 112. — Dominica (June—Oct.): Ibid., ii, 254; Clark (I). — Antigua, St. Kitts (June): Chisholm ii, 281, 291. — Santa Cruz (August—Oct.): Ibid., 342. — St. Thomas (November): Ibid., 320. — San Domingo: Moreau (I), 73. — Jamaica (June and fol.): Lemprière ii, 22, Rule. — Surinam: Stillé. — Philadelphia (August—Dec.): Rush (I), Curly, Currie (II, III), 'Facts and Observations,' Devèze (II), Pascalis (I), La Roche (II) i, 64.
1794 Demerara: Chisholm ii, 201. — Vera Cruz — Grenada (February): Chisholm i, 136; Stewart. — Dominica (July—Dec.): Clark (I), 5. — Martinique (March): Pym, p. 11; Gilpin (I). St. Thomas, Santa Cruz: Chisholm ii, 320, 342.—Havana (June—August.): Romay, Holliday. — Jamaica (June—August.): Lemprière ii, 52. — Charleston (July—Nov.): Ramsay (I), 36, Johnson (I). — Baltimore: Valentin (II), 71; Drysdale. — Philadelphia (August: Rush (II). — New York: Rush (II), 218. — New Haven (June—Nov.): Monson, Webster i, 302; ii, 340.
1795 Demerara: Eymann. — Grenada: Chisholm i, 138; ii, 231; Stewart. — St. Vincent, St. Thomas: Chisholm ii, 154, 320. —Martinique (January): Gillespie (I) 11. — Guadeloupe: Bishopp in Pym 118. — Domingo: Moreau (I) 74. — Jamaica (June—August): Walker, Lemprière ii, 22, 55, Todd. — Norfolk (August—Oct.): Valentin (I), 85; Taylor, Ramsay (III). — New York (July): Smith (VI), Seaman (I), Bayley (I), Hosack (I). — New London (August to Sept.): Monson.
1796 Demerara: Chisholm ii, 282; Beane. — Grenada (Summer—Autumn): Chisholm i, 138; Trotter ii, 83. — St. Lucia (June — July): Pym, Bally (I) 66. — Martinique (June—Sept.): Chisholm ii, 119; Davidson (I).—Dominica: Clark. — Tortola (July): Chisholm ii, 315; Anderson (I), 21. — Santa Cruz: Chisholm ii, 340. — Domingo: Bally (I), 65; Dalmas 33. — Jamaica (June—August): Lemprière ii, 55. — Bermuda: Barrow, Smart.—Charleston: Ramsay (I), 37; Johnson (I).—New Orleans: Thomas (I), 70; Chabert (I), 108. —New York (July—Dec.): Bayley (II), Webster i, 316.—

Wilmington, N. Car. (August—Nov.): Rosset.—Boston (August—Dec.): Warren (II).—Newbury-port, Mass. (June—Oct.): Coffin, Webster i, 318.—Chatham, Conn. (Sept.): Webster i, 331; ii, 344.

1797 Guayra, Venez.: Humboldt.—Trinidad (April): Fiedler.—St. Lucia: Gillespie 17.—Charleston: Ramsay (I), 37; Johnson (I).—Norfolk (August—Nov.): Valentin (I) 87.—Baltimore (June—Nov.): Davidge, Potter 52, New York Med. Repos. i, 380.—Philadelphia (July—Oct.): Devèze (I), Currie (I), Ouvière, 'Proofs;' Harris (I), 'Facts,' 64; Rush (III), Pascalis (II), Folwell.—Milesborough, Penns.: Harris (I).—Swedesborough, Penns.: 'Facts' 73.—Providence (August—Sept.): Wheaton, Bowen (I).—Bristol and other places in Rhode Island: Brown (II), Webster. i, 330.

1798 Domingo: Gilbert 137, 'Facts' 25.—Petersburg, Virg.: Erdmann 49.—Baltimore: Davidge 88, Potter 52.—Philadelphia (July—Oct.): Condie (I, II), 'Facts;' Currie (I, IV), Erdmann, 'Proceedings;' Caldwell (I).—Milesborough, Penns.: Harris (I).—Germantown, Penns.: Wistar in Hosack (III), 'Addit. Facts' 73.—Swedesborough, Penns. ib.—Chester, Penns.: New Y. Med. Repos. ii, 196; Erdmann 47.—Wilmington, Del. (August—Nov.): Vaughan (I), Erdmann 47, Monro, Tilton.—New-York (August—Nov.): Hardie (I), Hosack (II), M'Knight, Webster i, 334.—Bridgetown, Woodbury, Chews, N.-J. (sporadic cases): N. Y. Med. Repos. ii, 302; Erdmann 47.—Boston (June—Oct.): Brown (I, III), Rand, Webster i, 336; New Engl. J. of Med. viii, 380.—New-London, Conn. (August—Oct.): Cain, Channing, Holt, Scott, Webster i, 338.—Portsmouth, N. Hamp. (July): Erdmann 46, Webster i, 337.—Newport, Rh. Is.: Erdmann 47.

1799 New-Orleans: Chabert (I), 108; Carpenter 14.—Charleston (June to Oct.): Ramsay (IV).—Wilmington, Washington, N. Carol. (only sporadic), Norfolk, Baltimore (August—Oct.): New-York Med. Repos. iv, 197, 207.—Philadelphia (June—Oct.): Currie (V), Caldwell (I).—Swedesborough (sporadic cases): 'Addit. Facts' 74.—New-York, Boston, Providence, Rh. Isl. (only sporadic): N. Y. Med. Repos. l. c.—Hartford, Conn.: Webster i, 347.

1800 Demerara (August): Chisholm.—S. Domingo, Santa Cruz: Moreau (I), 172.—Surinam: Stillé.—New Orleans: Barton (I, untrustworthy account).—Charleston (July—Oct.): Ramsay (II).—Savannah (a few cases on board a vessel): Kollock.—Norfolk (July to Oct.): Hansford, Selden (I).—Wilmington (a few cases among sailors): N. Y. Med. Rep. iv, 319.—Baltimore: Moores, Chatard (I).—New York (Summer and Autumn): Seaman (II), N. Y. Med. Repos. iv, 207.—Providence (Aug. to Oct.): Wheaton.

1801 Martinique: Davidson (II).—St. Martin (July—Nov., seemingly for the first time): Dickinson (II), 179.—Jamaica (August to January): Doughty 4.—Norfolk (August—Oct.): Selden (II), New York (Summer and Autumn): New York Med. Repos. iv, 207.—New Bedford, Mass. (sporadic cases): Ibid. v, 275.—Block-Island, Rh. Isl., (August—Nov.) Willey.

1802	Caracas: Humboldt ii, 401.—Porto-Cabello: Moreau (I), 156.—Cayenne (Sept.): Leblond 226.—Vera-Cruz (April—Oct.): Bally (I) 78.—Tabago: Bouneau, p. 461.—St. Lucia (Aug. ff.): Pugnet 327.—Martinique (Sept.—Dec.): Moreau (I).—Guadeloupe: Lefonlon, Rouvier.—Antigua: Musgrave.—Santa Cruz: Keutsch (I).—St. Domingo (April—Nov.): Bally (I), 78; Gilbert, Dalmas, Beguerie, Mouillé, p. 10.—Jamaica: Doughty 16, Pym 184.—Charleston (August—Oct.): Ramsay (V).—Philadelphia (July—Nov.): Currie (VI).—Baltimore: La Roche (II) i, 93.—Wilmington, Del. (Sept.): Vaughan (II).—Boston (August—Oct.): N. Y. Med. Repos. vi, 338, in New-Engl. J. of Med. viii, 381.—Portsmouth, N Hampsh. (a few cases), ibid.
1803	Cayenne: Nogen.—Demerara: Frost.—Berbice (January—Sept.): Croissant in Thuessink, p. 117.—Martinique (the whole year): Moreau (I).—Guadeloupe: Bally (I), 81; Savarésy.—Santa Cruz (the whole year): Keutsch (II).—St. Domingo (to Nov.): Bally (I), 81; François (I).—Jamaica: Dancer, Rude.—Alexandria, Virg. (July ff.): Dick, N. Y. Med. Repos. vii, 177.—Baltimore: Ibid., vi, 235.—Philadelphia (July to Oct.): Caldwell (II), 17; Rush (III).—New-York (July to Oct.): Ramsay (VI), Mitchell (II), Stringham.
1804	Vera Cruz: Bally (I), 85.—New Orleans: Carpenter 17, Drake ii, 203.—Charleston (July—Oct.): Ramsay (VII).
1805	Martinique: Moreau (I) 118.—Santa Cruz (May—July): Keutsch (III).—Jamaica (Aug.—Jan.): Doughty 56.—Philadelphia (July ff.): Caldwell. (II); Rush (III), 96; 'Addit. Facts' 85.—New York (June to Nov.): Miller (I).—Providence (July—August): Wheaton, Bowen (II).—Newhaven: La Roche (II) i, 97.—Quebec (untrustworthy account): Moreau (I).
1807	Martinique (December—March, 1808): Moreau. (II).—Jamaica (Autumn): Doughty, p. 184.—Charleston (Aug. ff.): Ramsay (VIII), Johnson (II).
1808	Jamaica (Oct. ff.): Pym, p. 67.—Maria Galante (July—Sept.): Mortimer.—St. Marie, Geo. (September ff.); Seagrove.—Baltimore (August): Potter 21.
1809	St. Bartholomew (June—Nov.): Forström.—New Orleans: Carpenter 17; Drake ii, 203.—Brooklyn, N. J. (July): Gillespie (II).
1811	New-Orleans: Carpenter 17, Drake ii, 204.—St. Francisville, La.: Id., ii, 253.—Pensacola: Id., ii, 227.—Perth Amboy (September): Chisholm (III), Amer. Med. and Philos. Regist., iii, 94.
1812	Bermuda: Barrow, Smart, Jones, p. 204.
1813—14	Barbadoes: Thomas (III).
1815	Barbadoes: Fergusson (IV).
1816	Grenada: Dickinson (II), 48.—Guadeloupe: Musgrave (I), Vatable, Flory.—Martinique: Rochoux (II) 299.—St. Kitts (March ff.): Birnie.—Barbadoes (September—Feb. 1817): Ralph, Fergusson (IV), 593.—Antigua (June—Feb. 1817): Musgrave.
1816—18	St. Thomas (September 1816 to January 1818, epidemic): Gartner.
1817	Trinidad (August ff.): McCabe.—New Orleans (July—Oct.): Carpenter 17, Gerardin, p. 56; Gros et Gerardin.—St. Fran-

cisville: Carpenter 17, Drake ii, 253.—Baton Rouge: Id., ii, 250.—Natchez (July—Sept.): Perlee, Monette (II), Drake ii, 263.—Savannah: Daniell (I), 12, Posey.—Charleston (July—Nov.): Dickson (II), Shecut 100.

1818 Demerara: Lancet 1867, ii, 200.—Martinique (July (ff.): Lefort (also New Y. Med. Repos. xi, 350).—Bermuda (Sept. ff.): Jones, Barrow, Smart.

1819 Angostura, Venez. (July—Aug.): Morgan (I).—Demerara: Blair (I).—Martinique (Spring): Lefort.—Jamaica (May—June): Miller (II).—New Orleans (May—Dec.): Dupuy, Baxter, Fortin, Carpenter 18.—Baton Rouge: Drake ii, 250. —Natchez (September to Nov.): Perlee, Monette, Drake ii, 265.—Mobile (June to Dec.): Report (II), Drake ii, 217; Lewis (II).—Savannah: Watts, Amer. Med. Record, iii, 212.—Charleston (August): Shecut l. c., Irvine.—Baltimore (June—Sept.): Reese, Revere, 'Letters,' Chatard (II), Jameson.—Philadelphia (very slight epidemic): Emlen.—New York (August—Oct.): Drake (II), Pascalis (III), Brown (IV), Amer. Med. Record iii, 203 (also very limited diffusion).—Boston (August ff.): Report (I), Ingalis, New Engl. Journ. of Med. viii, 380.

1820 Demerara: Lancet, 1867, ii, 200.—New Orleans (July—Oct.): Carpenter, p. 19, Report (VIII).—Bay of St. Louis, Miss. (August): Merril (I), Report (VII) by Forry, p. 20.—Mobile (July to Dec.): Report (II).—Savannah (July): Fürth, Daniell (I), Posey, Arnold (II), Waring.—Philadelphia (July Nov.): Jackson (I), Report (IX).—Middletown, Conn.: Beck.

1821 Demerara: Lancet, l. c.—Martinique, Guadeloupe: Keraudren, p. 14.—Santa Cruz (April—July): Schlegel.—Tampico: Heinemann (II).—S. Augustine, Flor. (August—Dec.): Bayley (III), p. 248, according to Francis, Report (VII) by Forry, p. 30.—Savannah: Posey.—Wilmington, N. Carol. (July): Hill.—Norfolk (August to Oct.): Archer.

1822 St. Vincent: Hunter (II).—New Orleans (September—Nov.): Carpenter, p. 23; Randolph, Chabert (I, II), Thomas (I).—Baton Rouge: Drake ii, 250.—Pensacola (July—Nov.): Report (VII) by Forry 35, Drake ii, 228; Townsend (I).—New-York (July—Nov.): Bayley (III), Watters, Hardie (II), History (I), Yeates, Townsend (I, II).

1823 Jamaica (Summer and Autumn): Belcher.—Natchez (August): Drake ii, 266; Moneke, p. 66; Cartwright (I), Merrill (II), Tooley.—Key West (August—Sept.): Morgan (II), Ticknor.

1824 Jamaica (August ff.): Wilson (I). — New Orleans (July ff.): Carpenter, p. 23.—Charleston (Summer): Simons (I).

1825 Demerara: Lancet, l.c. — Guadeloupe: Chambolle. — St. Thomas (Feb.—May): Barclay. — Mobile (September—Oct.): Lewis (I), p. 286.—Natchez (Aug.—Nov.): Merrill (III), Cartwright (I).—Washington (Sept.): Monette (I).

1826 Guadeloupe, Martinique (Spring): Moreau (IV).

1827 Demerara: Lancet, l. c. — New Orleans (July): Drake ii, 207; St. Francisville: Id., 253.—Mobile (August): Id., 219. — Pensacola (Summer): Id. 230, Report (VII), by Forry. 58. — Savannah: Posey. — Charleston (August): Dickson (IV), Simons (I), Porter (II), 1855, April.

1828 Demerara: Lancet, l. c.—Martinique (October—Jan.): Moreau (V), Rufz (II). — New Orleans (June ff.): Drake ii, 207. —Memphis (September—Nov.): Id., 283.

1829 Havana, Jamaica (April—July): Moreau (VI). — New Orleans (July ff.): Id., Monett (IV). — Plaquemines (August): Drake ii, 249. — Francisville (Sept.): Id., 253. — Baton Rouge: Carpenter, p. 26. — Natchez: Monette (IV), Carpenter, l. c., Drake ii, 273. — Mobile: Id., 220, Lewis (I), l. c. — Key West (June—August): Dupré.

1831 Demerara: Lancet, l. c.

1833 St. Thomas (Feb.—May): Dons. — New Orleans (July—Nov.): Barton (I), Harris (II).

1834 New Orleans (August): Drake ii, 208. — Pensacola (August: Id., 232; Barrington.

1835 Surinam (Dec. ff.): Fraser (I), Pop, Dumortier (I). —Antigua (Sept.—Dec.): Furlong (I), Nicholson.

1836 Tampico, Mex. (Sept.—Nov.): Goupilleau.

1837 Demerara, Surinam (May—July): Fraser (I), Blair (I). — Havana: Maës.—New Orleans (July—Oct.): Thomas (II). —Opelousas (Oct.—Nov.): Carpenter, p. 52; Cooke (I).— Natchez (Sept. to Nov.): Monette (II), p. 75; Hogg. — Mobile (Sept.—Nov.): Lewis (I), 287; Nott (II), Drake ii, 220. — Bermuda: Barrow, Smart.

1838 Demerara: Blair (I), Lancet, l. c. — Trinidad (June). — Guadeloupe (July). — Martinique (Sept.—Oct., 1839): Catel, Rufz (I, II). — Dominica (April — June): Imray (I). — Charleston (July to Oct.): Wurdeman, Strobel, Hume (II), Simmons (II), Johnson (I).

1839 Demerara: ll. cc. — St. Vincent: Hunter (II). — Antigua (June): Nicholson. —Galveston, Houston (Sept.—Nov.): Smith (III, IV), Drake ii, 237. — New Orleans (August): Carpenter, p. 27, Lemoine, Journ. de la Soc. méd. de la New Orleans, 1839, Nr. 4. — Franklin, New Iberia (Sept.— Nov.): Carpenter, p. 27; 28, Monette (III), p. 111, 113; Drake, ii, 238, 241. — Opelousas (September): Carpenter, p. 59, Cooke (I). — Alexandria (September): Monette (III), l. c.— Natchitochez, Donaldsonville, Port Hudson, Waterloo, St. Francisville, Fort Adams (Sept): Monette (III), p. 108, 95, 98; Carpenter, p. 27; Drake ii, 254, 262. — Natchez (August): Id., ii, 275. — Bay of Biloxi: Monette (III), 117. — Mobile (August—Oct.): Lewis (I) 289. — Pensacola (Sept.): Carpenter, p. 27; Drake ii, 233. — Augusta (July ff.): Robertson, Strobel, p. 187. — Charleston (June ff.): Strobel, l. c., Hume (II), Simons (II).

1841 Demerara, Surinam: ll. cc., Dumortier (I).—Curaçao (Oct.— Nov.): Schorrenberg, Hommel.—Dominica (June—Sept.): Imray (II).—New Orleans (August—Oct.): Carpenter 29, Thomas (V), Barton (III).—Vicksburg (August—Nov.): Drake ii, 281.—Pensacola (August): Hulse, Laurison.— Key West (June to August): Dupré.

1842—45 Demerara: Blair (I).

1842 Antigua (Sept.—Nov.): Nicholson.—New Orleans (July): Carpenter, 29, 30.—Opelousas: Id., Cooke (I).—Mobile (August): Lewis (I), 289: Drake ii. 222.

1843 Galveston, Houston: Smith (IV).—New Orleans (July), St.

Francisville (August), Baton Rouge (October): Carpenter, p. 30, Drake ii, 251, 255.—Rodney, Miss. (September): Id., ii, 278; Williams (II).—Mobile (August—Dec.): Lewis (I), 290,—Charleston (September): Hume (II).—New York: v. Hohenberg.—Bermuda (July—Dec.): Report (VI), 1853, 176; Laird, Smart, Barrow.

1844 Guadeloupe: Carpentin, 45.—New Orleans: N. O. Med. J., i, Nr. 4.—Woodville (July—Sept.): Valetti, Stone (II).—Mobile: N. O. Med. J., l. c.

1845 Tamaulipas (Mexico): Heinemann (II).

1847 Barbadoes (Oct.—Dec., 48): Davy.—Vera Cruz (May ff.) und Tamaulipas: Porter (I), Heinemann (II).—Galveston, Houston: Smith (IV), Bracht, McCraven.—New Orleans (July—Oct.): Fenner (IV), Thomas (IV).—Covington, La.: Gilpin (II).—Rodney, Miss.: Williams (III).—Vicksburg: Hicks, Maegruder.—Natchez: Cartwright (II).—Mobile: Lewis (I), Nott (III).—Pascagoula (July—Nov.): Porter (III).

1848 New Orleans: Fenner (V).—Natchez: Stone (III).—New York (September): Bodinier.

1849 Antigua (Summer): Nicholson.—New Orleans (July—Dec.): Fenner, South. Med. Rep., i, 114.—Charleston (August—Nov.) Hume (IV), Simons (II).

1850 Cayenne (and next year): Kerhuel, Genouvês.—New Orleans (July—Oct.): Fenner (VI),

1851—53 Demerara (from Dec. 51 to Dec. 53): Lancet, l. c., Blair (II).—Paramaribo, Sur. (1851): Pop.

1852 Charleston (August—Nov.): Porter (II), Simons (II).

1852—53 Antilles in universal diffusion: St. Thomas (starting point of the epidemic, August—Jan.): Hildige, Archambault, 'Aarsberetning,' 1853, i, 35, 36.—Domingo: Ibid., ll. cc.—Guadeloupe (Dec. ff.): Dutroulau (III), Carpentin, l. c.—Martinique: Encognère, Rufz (II).—Santa Cruz (Oct.—April): 'Aarsberetning,' l. c.—Curaçao (Dec. to 1855 continuously): Pop, Nederl. Tijdschr. voor Geneesk, 1858, 223.—Cuba, Porto-Rico, Dominica, St. Lucia. Barbadoes: Hildige.

1853 Coast of Mexico (general diffusion): La Roche ii, 623; also in the Antilles: Ibid.—Antigua (May): Nicholson, Furlong (II).—Barbadoes: Teevān.—St. Thomas (April—August): 'Aarsberetning,' 1856, p. 68.—Gulf Coast of N. America and up the Mississippi Valley, in general prevalence: La Roche, l. c.—New Orleans (May—Oct.): Fenner (I), Mercier, Dowler (I), N. Orleans Med. J., 1853 Sept. Barton (II).—Madisonville, Mandeville, Louisbourg, Covington, Baton Rouge, Port Hudson, St. Francisville, Bayou Sara: Dowler (I).—Franklin, La.: Lyman.—Washington, La.: Cooke (II).—Arkansas in several localities on the Mississippi: Dowler (I).—Natchez: New Orleans Med. News and Gaz., 1855, Oct. 1.—Clinton, Jackson, Grand Gulf, Yazoo, Vicksburg, Miss.: Dowler, McAllister.—Memphis, Tenn.: Smith (VIII).—Mobile: Anderson (III), Nott (IV), Ketchum.—Selma, Ala.: Marks.—Florida (various points on the coast): Dowler.—Brandywine, Del. (July—Sept.): Bush.—Philadelphia (July—Oct.): Jewell (I), Bache, La Roche (I, II) i, 110.—Bermuda (August to Nov.): Barrow, Milroy, Smart.

1854	Surinam: Pop, Dumortier (I).—Curaçao (Febr.): Landré.—Antigua: Morehead.—Galveston, New Orleans (July ff.): Med. News.—Mobile (August—Nov.): Ketchum.—Montgomery, Ala., St. Marie, Darien, Augusta, Geo.: Med. News.—Savannah, Geo.: Posey, Hume (V), MacKall, Arnold (II).—Charleston: Cain, Chisholm, Hume (III).
1855	Cayenne (May ff.): Kerhuel.—Martinique (Oct.—August, 1857): Ballot, Chapuis, Encognère, Pellarin.—St. Thomas (Jan.—Nov.), Santa Cruz (April ff.): 'Aarsberetning,' 1856, 71.—Domingo: Archambault.—Havana: Ramon.—New Orleans: Med. News.—Natchez: New Orl. Med. News and Gaz., 1855, Oct. 1.—Vicksburg, Yazoo, Cooper's Well, Jackson, Clinton, and other places in Mississippi, Memphis, Tenn.: Med. News.—Portsmouth and Norfolk (July—Oct.): Bryant, Fenner (II), Williman, Report (III), Warren (III).
1856	Cayenne (Jan. ff.): Kerhuel.—Martinique, Guadeloupe, St. Thomas: 'Aarsberetning,' 1857, 297.—Jamaica: Lawson, Brit. Army Reports for 1867, ix, 216.—New Orleans (July ff.): New Orl. Med. Times, 1856, Sept.—Charleston (July—Sept.): Dawson, Charlest. Med. Journ., 1856, Dec., 845, Hume (VI).—New York (July—Sept.): Harris (IV), Med. News, 1856, 144, and N. York Journ. of Med., 1856, Sept., Buckley.—Long Island (July—Aug.): Buckley, Rothe.—Bermuda (July—Oct.): Barrow, Milroy, Smart.
1857	Martinique: Encognère.—Guadeloupe: Batby-Berquin.—St. Thomas (April—July), Santa Cruz (Sept.—Nov.): 'Aarsberetning,' 1858, 429.—St. Domingo: Archambault.—New Orleans: Mercier, 468.—Charleston (August—Sept.): Kinloch, Hume (VIII).—Jacksonville, Flor. (Oct.): Porcher.
1858	Panama.—Antigua (Dec. ff.).—St. Thomas (April, Nov.—Dec.): 'Aarsberetning,' 1859, 436.—Trinidad (Sept.).—Matamoros: Heinemann (II).—Galveston.—Brownsville, Tex. (August—Oct.): Watson.—New Orleans (June—Oct.): Faget, 32, Mercier, 475, 567.—Mobile, Savannah.—Charleston (Sept. ff.): Report (IV), Charlest. Med. Journ., 1858, Nov., 841, Byrne, U.S. Army Reports, 1860, 122.—Philadelphia (Summer, a few imported cases): Jewell (II).
1859	Panama.—Trinidad.—St. Thomas (Oct.—Dec.): 'Aarsberetning,' 1860, 409.—Curaçao (Nov.—Feb., 60): Nederl. Tijdschr. voor Geneesk, 1860, 127, 256.—New Orleans (Sept. ff.); N. Orl. Med. News and Hosp. Gaz., 1859, Dec.—Texas: Dowler (III).
1860	Honduras (Belize): Hamilton.—Cuba, Jamaica, Domingo, Martinique, and others of the Windward Islands.
1861	Cartagena.—Nassau (Bahama).—Martinique.—St. Thomas.—Halifax, Nova Scotia (July—Sept.): Slayter, Mauger.
1861—66	Demerara (continuously from May, 1861 to 1866 mostly on board ship): Lancet, 1867, ii, 200, and in Brit. Army Reports for 1868, x, 69.
1862	Vera Cruz (March—Sept.): Crouillebois, Bouffier, Legris, Buez in Gaz. Méd. de Paris, 1862, 440.—Cuba.—Tortuga.—St. Lucia.—Barbadoes: McGregor in Brit. Army Reports for 1864, vi, 256.—Bahama: Ib., 255.—Louisiana (several places on the coast).—Key West (July): Horner, Med. History of the Rebellion, Philad., 1865, p. 113.—Pensacola.

	—South Carolina (several places on the coast), Hilton Head, S. Carol. (August): History of the Rebellion, ib.—Wilmington, N. Car.
1863	Vera Cruz, Matamoros, Tuxpam, Tampico, Mex.: Jaspard, Heinemann (II).—Nassau, Bah.—Pensacola (July—May, 1864): Gibbs.
1864	Bermuda (June—Dec.): Barrow, Report (V).—Newbern, N. Carol. (Summer): History, l. c.
1864—67	Vera Cruz: Mouffley, Schmidtlein.
1865	Guadeloupe.—Domingo: Dupont, p. 55.—Campeche (Mex.): Heinemann (II).
1866	Cayenne.—Paramaribo: Gori, Nederl. Tijdschr. voor Geneesk, 1866, i, 589.
1866—67	Jamaica: Donnet, Arch. de Med. nav., 1870, July, August; Brit. Army Rep. for 1867, ix, 224.—St. Thomas: Miller (III).
1867	Panama (Pacif. Coast).—Cuba (Summer): Dunlop.—Barbadoes and others of the Antilles: Med. News.—Matamoros: Heinemann (II).—Texas (Autumn) in Galveston, Corpus Christi, Indianola, Lavacca, Lagrange, Victoria, Huntsville, Hempstead, Mellican, Alleyton, Navasola, &c.: Kearney, New York Med. Record, 1867, Oct., 373; Heard in Transact. of the Amer. Med. Assoc., xix, Philad., 1868.—New Orleans (June—Nov.): Stone (I), Schmidt, New Orl. Med. Journ., 1867, Nov.; Chaillé, ib., 1870, Jan.—New Iberia, La.—Memphis: Mallory.—Vicksburg.—Mobile.—Key West (August ff.): Dunlop, Thompson.—Pensacola: Philad. Med. and Surg. Reporter, 1868, March, 227.
1867—68	Vera Cruz: Lancet, 1868, Aug., 268.
1868	Manzanillo, Mex.: Philad. Reporter, l. c.—Nicaragua, San Salvador: Guzman, Gonzalez in France Méd., 1869, No. 46. —St. Kitts: Munro.
1868—69	Martinique: Rufz (II), Bakewell, in Lancet, 1869, Dec., 794.
1868—70	Cuba (Havanna, Matanzas, Cienfuegos, Sagua, &c.), Philad. Reporter, l. c., Poggio in Siglo Medico (Med. Times and Gaz., 1869, Oct., 516), Sullivan in Med. Times and Gaz., 1871, March, 304 —Guadeloupe: Rufz (II), Batby-Berquin, Griffon du Bellay, Carpentin.
1869	Caracas, Venezuela.—Nassau, Bah.—Trinidad: Bakewell, l. c.
1870	Governor's Island, N. York: Sternberg.
1871	Charleston: Porcher in Transact. of the South Carolina Med. Assoc. for 1872, p. 3.
1873	Southern States (in general diffusion): Texas (Marshall, Calvert, &c.).—New Orleans: Jones (II).—Shreveport, La.: Jones (III), Marsh.—Memphis, Tenn.: Mallory.—Montgomery and other places in Alabama: Miehel in Transact. of the State Alabama Med. Association for 1874.—Vicksburg: Transact. of the Mississippi State Med. Assoc. for 1874.—Bainbridge, Geo., &c.
1874	Pensacola, Flor.
1875	Mexico (severe epidemic): Heinemann (II).
1876	Cordoba, Mex.: Heinemann (II).—Savannah, Brunswick, Geo.: Woodhull, White, Purse, Report (IX).—Baltimore.
1877	Jamaica: Med. Times and Gaz., 1877, Sept., 340.—Cayenne:

	Crevaux.—Mexico (severe epidemic): Heinemann (II).—Jacksonville. Flor.
1878	Mexico (severe epidemic): Id.—Southern States of the Union in wide diffusion: Jones (IV), Bayley (II), numerous accounts in New York Med. Record and Philad. Med. and Surg. Reporter, 1878.
1879	Memphis: Thornton.

Table according to Latitude of the Yellow Fever Epidemics in the West Indies, N. America, C. America, and the Gulf Coast of S. America.[1]

Guiana:
- *Cayenne (4°56 N.) 1763—65, 1791, 1802, 1803, 1850, 1855, 1856, 1866, 1877.
- *Surinam (5°50 N.) 1760, 1793, 1800, 1803, 1835, 1837, 1841, 1851, 1854, 1866.
- *Demerara (6°49 N.) 1793—96, 1800, 1803, 1818, 1819, 1820, 1821, 1825, 1827, 1828, 1831, 1837—39, 1841—45, 1851—53, 1861—66.

Venezuela:
- †Angostura (8°8 N.) 1819.
- *Guayra, Caracas (10°30 N.) 1693, 1696, 1793, 1797, 1802, 1869.
- *Porto Cabello (10°29 N.) 1802.

New Granada:
- *Cartagena (10°25 N.) 1740, 1861.

Central America:
- *Panama (8°57) 1740, 1858, 1859, 1867.
- *Portobelo (9°24) 1726, 1729, 1740, 1798, 1860, 1866, 1867.
- *Nicaragua (10°—15°) 1868.
- *San Salvador (13°—14°) 1868.
- *Belize (Honduras) (17°30) 1860.

Antilles in wide diffusion: 1699, 1860, 1867.
- *Trinidad (10°20) 1793, 1797, 1817, 1838, 1858, 1869.
- *Tabago (11°20) 1793, 1802.
- *Grenada (12°2) 1770, 1791, 1793—96, 1816.
- *Curaçoa (12°6) 1751, 1760, 1841, 1852—54, 1859.
- *St. Vincent (13°10) 1793, 1795, 1822, 1839.
- *Barbadoes (13°10) 1647, 1690, 1694—95, 1699, 1715, 1721—24, 1733—39, 1793, 1813—16, 1847, 1852—53, 1862, 1867.
- *St. Lucia (13°50) 1665, 1767, 1796—97, 1802, 1852—53, 1862.
- *Martinique (14°30) 1688—90, 1693—94, 1696—97, 1699, 1703, 1705—6, 1750, 1770—72, 1793—96, 1801—3, 1805, 1807, 1816, 1818—19, 1821, 1826—28, 1838, 1852—53, 1855—57, 1860, 1861, 1868—69.
- *Dominica (15°18) 1793—94, 1796, 1838, 1841, 1852—53.
- *Maria Galante (16°) 1808.
- *Guadeloupe (16°10) 1635—49, 1699, 1703, 1795, 1802—3, 1816, 1821, 1825—26, 1838, 1844, 1852—53, 1856—57, 1865, 1868—70.

[1] * Indicates places on the coast. † Places on large navigable rivers. ‡ Places in the interior on small rivers or at a distance from rivers.

*Antigua (17°8) 1729, 1754, 1756, 1765—66, 1793, 1802, 1816, 1835, 1839, 1842, 1849, 1853—54, 1858.
*St. Kitts (17°17) 1648, 1652, 1699, 1793, 1816, 1868.
*Santa Cruz (17°44) 1690, 1793—94, 1796, 1800, 1802—3, 1805, 1821, 1852—53, 1855, 1857.
*St. Bartholomew (17°55) 1809.
*St. Martin (18°5) 1801.
*St. Thomas (18°20) 1793—95, 1816—18, 1825, 1833, 1852—53, 1855—59, 1861, 1866—67.
*Tortola (18°27) 1796.
*Puerto Rico (17°—18°) 1852—53.
*San Domingo (17°—19°) 1656, 1690, 1696—97, 1699, 1705, 1731, 1733—37, 1740—41, 1743—46, 1781—82, 1793, 1795—96, 1798, 1800, 1802, 1803, 1852—53, 1855, 1857, 1860, 1862, 1865.
*Jamaica (17°—18°) 1655, 1671, 1741—42, 1780, 1793—96, 1801—3, 1805, 1807—8, 1819, 1823, 1824, 1829, 1856, 1860, 1866—68, 1877.
*Cuba (23°9) 1762, 1780—82, 1794, 1829, 1837, 1852—53, 1855, 1860, 1862, 1867—70.
*Nassau (Bahamas) (25°4) 1861, 1862, 1863, 1869.

Mexico:
*Vera Cruz (19°11) 1699, 1725, 1740, 1794, 1802, 1804, 1847, 1853, 1862—65, 1868, 1875, 1877, 1878.
*Manzanillo (19°12) 1868.
*Campeché (19°51) 1865, 1877.
*Tuxpam (22°10) 1863, 1875, 1877.
*Tampico (22°15) 1821, 1836, 1845, 1847, 1853, 1864.
*Papántla (22°15) 1877.
*Matamoros (20°25) 1858, 1863, 1867.
‡Cordoba (18°50) 1876.

North America:
Texas, 1859, 1867 (in many inland places) 1873:
*Galveston (29°18) 1839, 1843, 1847, 1854, 1858, 1859, 1867.
†Houston (30°) 1839, 1843, 1847, 1859, 1867.
*Corpus Christi (27°25) 1859, 1867.
†Indianola (29°20) 1859, 1867.
†Lagrange (30°25) 1859, 1867.
†Brownsville (25°54) 1858.

Louisiana (29°—31°60) in wide diffusion, 1862, 1873:
†New Orleans, 1791, 1796, 1799, 1800, 1804, 1809, 1811, 1817, 1819, 1820, 1822, 1824, 1827—29, 1833—34, 1837, 1839, 1841—44, 1847—50, 1853—59, 1867, 1873, 1878.
†Plaquemines, 1829, 1878.
*Madisonville, 1853.
†Louisburg, 1853.
†Mandeville, 1853.
†Covington, 1847, 1853.
†Franklin, 1839, 153.
†New Iberia, 1839. 1867.
†Donaldsonville, 182.
†Port Hudson, 1829, 1853.
†Waterloo, 1829.
†Baton Rouge, 1817, 1819, 1822. 1829, 1843, 1853, 1878.
*Port Eads, 1878.
‡Morgan, 1878.
‡Opelousas, 1837, 1839, 1842.
†St. Francisville, 1811, 1817, 1827, 1829, 1839, 1843, 1853.

†Alexandria, 1839.
†Natchitochez, 1839.
†Shreveport, 1873.
†Arkansas (33°—34°) at several places on the Mississippi, 1853.
Mississippi (30°28—32°50):
*Shieldsborough, 1820, 1829.
*Biloxi, 1702, 1839.
*Pascagoula, 1847.
*Port Gibson, 1878.
†Port Adams, 1839, 1853.
‡Woodville, 1844.
†Rodney, 1843, 1847.
†Natchez, 1817, 1819, 1823, 1825, 1829, 1837, 1839, 1847—48, 1853, 1855.
‡Washington, 1825.
†Grand Gulf, 1853.
†Jackson, 1853, 1855.
†Clinton, 1853, 1855.
†Vicksburg, 1841, 1847, 1853, 1855, 1867, 1873, 1878.
†Yazoo, 1853, 1855.
Tennessee:
†Memphis (35°5) 1828, 1853, 1855, 1867, 1873, 1878, 1879.
†Brownsville, 1878.
†Chattanooga, 1878.
Alabama (30°41—32°45):
*Mobile, 1765, 1819, 1820, 1825, 1827, 1829, 1837, 1839, 1842—44, 1847, 1853—54, 1858, 1867, 1878.
†Montgomery, 1854, 1873.
†Selma, 1853.
Florida (24°32—30°24) 1853, at many places on the coast.
*Pensacola (30°24) 1765, 1811, 1822, 1827, 1834, 1839, 1841, 1862, 1863, 1867, 1874.
*Key West (24°32) 1823, 1829, 1841, 1862, 1867.
*St. Augustine (29°60) 1821, 1841.
†Jacksonville (30°) 1857, 1877.
Georgia (30°75—33°45):
†St. Mary, 1808, 1854.
*Darien, 1854.
*Brunswick, 1876.
*Savannah, 1800, 1817, 1820—21, 1827, 1854, 1858, 1876.
†Augusta, 1839, 1854.
*Bainbridge, 1873.
South Carolina:
*Hilton Head (32°10) 1862.
*Charleston (32°46) 1693, 1699, 1700, 1728, 1732, 1739, 1745, 1748, 1792, 1794, 1796—97, 1799, 1800, 1802, 1804, 1807, 1817, 1819, 1824, 1827, 1838—39, 1843, 1849, 1852, 1854, 1856—58, 1862, 1871.
Bermudas (32°) 1699, 1779—80, 1796, 1812, 1818, 1837, 1843, 1853, 1856, 1864.
North Carolina:
*Wilmington (34°11) 1796, 1799, 1800, 1821, 1862.
*Newbern (35°20) 1864.
*Washington (35°40) 1799.
Virginia:
*Norfolk (36°50) 1737, 1741—42, 1795, 1797, 1799, 1800—1, 1821, 1855.

†Petersburg (37°13) 1798.
*Alexandria (38°49) 1803.
Maryland and Delaware:
*Baltimore (39°17) 1783, 1794, 1797—1800, 1802—3, 1808, 1819—22, 1876.
*Wilmington (39°41) 1798, 1802.
†Brandywine (39°43) 1853.
Pennsylvania (39°50—40°15):
*Marcus Hook, 1798.
†Philadelphia, 1693, 1699, 1741, 1747, 1762, 1793—94, 1797—99, 1802—5, 1819—20, 1853, 1858.
†Chester, 1798.
†Swedesborough, 1797—99.
†Milesborough, 1797—98.
†Germantown, 1798.
New Jersey (39°40—40°45):
*Bridgetown, 1798.
†Chews, 1798.
†Woodbury, 1798.
*Perth Amboy, 1811.
New York (40°41—40°42):
*New York, 1693, 1702, 1743, 1745, 1791, 1794—96, 1798—1801, 1803, 1805, 1819, 1822, 1843, 1848, 1856, 1870.
*Brooklyn, 1809.
*Long Island, 1856.
Connecticut (41°18—41°45):
*New Haven, 1743, 1794, 1805.
*New London, 1795, 1798.
†Middletown, 1820.
†Chatham, 1796.
†Hartford, 1799.
Rhode Island (41°13—41°49):
*Block Island, 1801.
*Newport, 1798.
*Bristol, 1797.
*Providence, 1797, 1799, 1800, 1805.
Massachusetts (41°38—42°48):
*New Bedford, 1801.
*Boston, 1796, 1798—99, 1802, 1819.
*Newburyport, 1796.
New Hampshire:
*Portsmouth (43°4), 1798, 1802.
Canada:
†Quebec (46°50) 1805.
Nova Scotia:
*Halifax (44°26) 1861.

On the mainland of South America yellow fever has become widely diffused only since the sixth decade of the present century. Whether the information about malignant sickness in Pernambuco in 1640, 1687, 1710, and 1780, relates, as McKinlay believes, to yellow fever, is at least doubtful;[1] still

[1] The description given by Piso ('De medicina Brasil.,' lib. i, p. 13; lib. ii, p. 15) in the middle of the 17th century of a pernicious fever observed in

less credit is due the notion that the epidemic of 1647 in Santiago[1] and that of 1781 in Lima[2] were yellow fever. Thus during the whole of the past, up to the year 1850, there have been only *two* epidemics observed on South American soil that may be designated with certainty as yellow fever. Both of these happened at Guayaquil; the first in 1740, concerning which an importation of the disease from outside is expressly mentioned;[3] and the second in 1842, which can in like manner be proved to have been introduced by strangers who had come from New Orleans by way of Panama.[4]

The general outbreak of yellow fever in Brazil dates from the end of 1849;[5] in October the sickness appeared in Bahia, having been imported either from New Orleans or Havana; it soon attained the extent of an epidemic, and from Bahia, Rio Janiero was infected in December, Pernambuco in February, 1850, and Para, Alagoes, and Parahiba, almost at the same time, while Santa Catarina and Santos were attacked in March. In the summer of the following year the disease first showed itself in St. Louis de Maranhao, and in June it was at Ceara, so that by that time the whole coast and the country for several miles inland was overrun by the pestilence. In 1852-54 the disease was prevalent in epidemic form at isolated points only, in 1855 it again broke out over a wide area, and it spread in the following year along the Amazon far into the interior of the country.[6] Since then it has never left Brazil altogether; its years of special severity have been 1859-60, 1862, 1869-70, 1872-73, and 1875-77.[7]

From Brazil yellow fever came first to *Peru* in 1854, and it was probably brought by a vessel with German emigrants.[8] The

Brazil does not suit yellow fever; and as regards the epidemic described by Ferreyra da Rosa ('Trattado da constituiçao pestilencial de Pernambuco,' Lisboa, 1694), which was prevalent in Pernambuco from 1687 to 1694, Sigaud (p. 116) expressly states that it had nothing in common with yellow fever.

[1] Frezier, 'Relat. du Voyage dans le Mer du Sud, &c.,' Paris, 1716, p. 41.

[2] Leblond, p. 190.

[3] Ulloa, p. 149.

[4] Celle, p. 80; Smith (VII), p. 244.

[5] See McWilliam, Baker, Paterson, M'Kinlay, Döllinger, Wucherer, Lallemant, and especially De Moussy, 'Description géogr. et statist. de la Confédération Argentine.'

[6] Lallemant, p. 120 ff.

[7] Rey, following the numerous reports of Brazilian physicians.

[8] See Eysaguirre. The statements of Smith (I and V) about the yellow fever in Peru are to be received with caution, inasmuch as that author has confounded the disease with the dengue which was prevalent there at the same time, as well as with a febrile sickness which occurred afterwards in the

disease appeared first in the port of Callao, shortly thereafter in Lima. In the years following it spread over a great part of the Peruvian coast, without, however, penetrating into the mountainous regions of the interior. From the first appearance of the sickness in 1854 down to 1868-69, when it raged with unusual severity,[1] yellow fever had never been absent from Peru.[2] Up to the present time *Chili* has remained free from yellow fever; on the other hand, since 1857 the disease has been several times introduced from Brazil into the *States of the River Plate*, first into Monte Video[3] in 1857, next year into Buenos Ayres,[4] in 1869 to Asuncion, where it was prevalent also the year after,[5] further into Corrientes[6] and Buenos Ayres[7] in 1870, and again into Monte Video from Pernambuco in 1872.[8] Since that date yellow fever does not appear to have visited those regions.

A striking contrast to the large area of distribution of yellow fever in the Western Hemisphere is presented by the very limited occurrence of the disease in the Old World, where, if we disregard the quite isolated outbreaks in the South-West of Europe, it is met with at one point only, viz. the *West Coast of Africa*. The records of physicians and travellers in that region in former centuries are too scanty and unreliable to enable us to decide how soon yellow fever showed itself after the first settlements of Europeans on the coast. The earliest trustworthy information[9] is to be found in the account by Schotte of the epidemic of yellow fever at St. Louis (Senegal) in 1778, an epidemic which, like all the later ones at the same place, could be traced to an importation of the disease from *Sierra Leone*. The latter strip of

mountainous parts of the country, and was proved by Macedo ('Gaz. med. de Lima,' 1858, No. 48) and others to have been not yellow fever, but typhus.

[1] Account in the 'Lancet,' 1869, March, p. 446.

[2] Boilleau, in the 'Compt rend.,' 1869, lxix, No. 18.

[3] Brunel, 'Mantegazza,' i, p. 10, Scrivener (II).

[4] Scrivener (I).

[5] Hiron.

[6] Id.

[7] Id., Scrivener (I), Leeson.

[8] Scrivener (II), Brendel.

[9] Busto y Blanco mentions two epidemics of yellow fever in the Canary Island, 1701 and 1771, introduced, however, in both cases, as we are told, from the West Indies.

coast appears, indeed, to be the headquarters of the disease, and the starting-point of its epidemic inroads into the territories lying to the north and south, as well as into the West African Islands. In the two following tables I have put together, in chronological order and according to latitude respectively, the yellow fever epidemics that are known to have occurred over the whole of the territory here mentioned since the beginning of this century.

Chronological survey of yellow fever epidemics on the West Coast of Africa and the West African Islands.

1810	Teneriffe (introduced from Cadiz): Busto y Blanco (I), Vergoara.
1816	Sierra Leone: Bancroft ('Sequel,' p. 240).—Congo Coast: Moreau (XIV).
1823	Sierra Leone: McDiarmid, p. 448; Boyle, p. 201; Bryson (I), p. 35, Gore, p. 405.—Ascension: Burnett (I), Fergusson (II), 840.
1825	Sierra Leone: Boyle, p. 201; Gore, p. 407.
1829—30	Sierra Leone: Boyle, Bryson, McDiarmid, ll. cc.; Gore, p. 408.
1830	Senegambia: Thevenot, p. 254; François (II), Chevée.
1837—39	Sierra Leone: Bryson (I), p. 65; McDiarmid, p. 444, Clarke, Gore, p. 409.
1837	Senegambia: Fergusson (III), p. 841; Cedont.—Ascension: same references as under 1823.
1839	Fernando Po: Bryson (I), p. 68.
1845—47	Sierra Leone: Bryson (I), p. 156; Clarke, Gore, p. 411.
1845—46	Boa Vista (Cape Verd Islands): McWilliam, King, Bryson (I), p. 96.
1846	Canary Islands (imported from America): Busto y Blanco.
1852 1857	Gold Coast (Grand Bassam, Dabon): Sarrouille.
1858	Senegambia (Gorée).
1859	Sierra Leone: Clarke, Gore, p. 412.—Senegambia (Gorée, Bathurst): Cedont.
1860	Gambia (Macarthy's Island).—Congo Coast (Loanda, Angola).
1862	Sierra Leone.—Gold Coast: Sarrouille.—Benin Coast (Calabar): Account in 'Transact. Lond. Epidemiol. Soc.,' i, 387.—Congo Coast: Ref., ib.—Fernando Po.—Cape Verd Islands.—Teneriffe and Palmas (Canary Islands): Busto y Blanco.
1864	Sierra Leone (Lagos).
1865	Sierra Leone: Gore, p. 413; Mackay.—Congo Coast (Loanda): Mackay.
1866	Sierra Leone: ll. cc.—Senegambia (Gorée, Bathurst): Cedont.
1867	Senegambia (St. Louis): Carbonnel, p. 30.
1868	Sierra Leone.—Senegambia.—Cape Verd Islands (St. Jago, Brava): De Silva.
1878	Sierra Leone (?)—Senegambia: Crevaux.

Chorographic survey of the epidemics of yellow fever on the West Coast of Africa and the West African Islands.

Sierra Leone: 1816, 1823, 1825, 1829—30, 1837—39, 1845—47, 1859, 1862, 1864, 1865—66, 1868, 1878 (?).
Senegambia: 1778, 1830, 1837, 1858, 1859, 1860, 1866, 1867, 1868, 1878.
Gold Coast: 1852, 1857, 1862.
Benin Coast: 1862.
Congo Coast: 1816, 1860, 1862, 1865.
Ascension: 1823, 1837.
Fernando Po: 1839, 1862.
Cape Verd Islands: 1845, 1862, 1868.
Canary Islands: 1701; 1771, 1810, 1846, 1862.

On the north coast of Africa, yellow fever has been observed only once, in 1804, on the small island containing the Spanish fortress of Alhuzemas, lying off the coast of Morocco, whither it had been imported from Catalonia.[1]

The farthest point hitherto to which yellow fever has extended its epidemic range is the South West of *Europe;* and if we set aside an occurrence of it once (1694) at Rochefort (where the fever might be regarded as pernicious malarial[2]), and once (1804) at Leghorn (whither it was imported from Cadiz[3]), its European area is limited to the *south-west coast of the Iberian peninsula and to Majorca.*

The series of yellow fever pestilences in *Spain* begins with the year 1700, when the disease appeared as an epidemic at Cadiz,[4] without, however, spreading to other places; that was also the case with the subsequent epidemics in that town in the years 1730-31,[5] 1733-34,[6] 1764,[7] and 1780.[8] So that if we except a Lisbon epidemic of yellow fever in 1723,[9] and a Malaga epidemic in 1741,[10] Cadiz remains the only place in the peninsula that was visited by the disease during the eighteenth century. With the commencement of the present century the sickness

[1] Bally (II).
[2] Chirac, 'Traité des fièvres malignes,' Paris, 1742, p. 30.
[3] Palloni, Lacoste, Barzelotti, Dufour.
[4] Arejula (II), p. 454.
[5] Fellowes, p. 23, Gonzales, p. 5, Villalba, ii, 185.
[6] Bally (I), p. 42.
[7] Fellowes, p. 25, Gonzales, p. 6.
[8] Ketterling.
[9] Sanchez, Bancroft (I), p. 436 (from a MS. account by Kennedy), Lyons, pp. 8, 110.
[10] Rexano, Rubio, Villalba, ii, 203; see also Martinez y Montes, 'Topogr. med. de la Ciudad de Malaga,' Mal., 1852, p. 486.

in Spain reached more considerable dimensions, spreading in the years 1800—1804 from Cadiz, which was again first attacked, in epidemic form over a great part of Granada and Andalusia, northwards along the banks of the Guadalquiver to Cordova, thereafter deep into the interior, and from Andalusia to the seaboard of Murcia, Valencia, and Catalonia.[1] We meet with a second but more limited epidemic of yellow fever on Spanish soil in 1810; the disease broke out in the autumn almost simultaneously at Cadiz,[2] Cartagena,[3] and Gibraltar,[4] in the two following years it appeared at the same places anew, and from them it again spread through several of the coast towns of Granada, Murcia, and Valencia.[5] The third and last great epidemic of yellow fever on Spanish soil was in the years 1819—21; this time also it was Andalusia and Granada that suffered most, afterwards also Murcia and Catalonia, in which not only Barcelona and other places on the coast were attacked, but also several towns in the interior, for example, Tortosa.[6] From Barcelona the disease was imported in 1821, as it had been in 1804,[7] into Palma in Majorca.[8] Since 1821 yellow fever has reappeared as an epidemic four times in Spain, but always within narrow limits; it was imported in 1823 into the small port of Los Passages (on the Bay of Biscay) by a Spanish brigantine from Havana;[9] into Gibraltar[10] in 1828, and into Barcelona[11] in 1870, both times from the West Indies; and on the last occasion it was carried by the shipping

[1] See the writings of De Maria, p. 23, Bally (I), pp. 71—90, Salgado, Gonzalez, Arejula (I) and (IV), p. 137 ff—308, Armesto, Fellowes, pp. 33, 87, Ferrari, Berthe *passim*, Kerandren (II), Salamanca, Pym, p. 20, Souerampe; further, the epidemiological reports in the 'Period. de la Sociedad med.-quir. de Cadiz,' 1824, tom. iii, No. iv, Append., the 'Discorso sobre el Origin . . . . de la Enfermedad malign. cont. en la Ciudad de Cadix, 1800,' Cadiz, 1800, the 'Manifesto sobre la Epidemia . . . de Sevilla . . . en año 1800, &c., Sevilla, 1800, and in the 'British Army Reports,' 1839, 10 *a*.

[2] Doughty, Fellowes, pp. 226, 287, Leiblin, Flores, Melado.

[3] De Maria, p. 123, Vance, Proudfoot.

[4] Burnett (II), Pym, p. 47, Amiel (I), Gilpin (I), Humphrey.

[5] See Fellowes, pp. 238-39, Paris-et Mazet, p. 75, Velasquez, Vance, Pym, p. 61.

[6] Pariset et Mazet, Ferrari, Jackson (III), 9, 64, O'Halloran, Bally (II), Audouard (I), Rochoux, Bahi, Costa, 'Rapport . . . . sur l'origine de la fièvre jaune, qui régne en 1821 à Barcelona, &c.,' trad. par Rayer, Paris, 1822; Account in the 'Report général d'anat. et de physiol. pathol.,' 1826, I, 1, and numerous notices in the 'Period. de la Soc. med.-quir. de Cadiz,' 1824, l. c.

[7] Bally (II), p. 61.

[8] Id., p. 64, Almedovar.

[9] De Arruti, Montes, Collineau, Audouard (II), Melier, p. 301.

[10] Louis, Chervin (I), Wilson (II), Guyon (I), Amiel (II), Barry, Fraser (II).

[11] 'Memoria historico-cientifica sobre la epidemia de febre amarilla sufrida en Barcelona en 1870, &c.,' Barcelona, 1872; Larrey; see also Ullersperger, in the 'Bayr. ärztl. Intelligenzbl.,' 1870, No. 44, and the 'Deutsche Klinik,' 1871, No. 15.

traffic to Alicante, Valencia, and Palma (Majorca).[1] Finally it came in 1878 to Madrid along with troops arriving from Cuba,[2] as will be afterwards mentioned.

From 1723, when there was an epidemic at Lisbon, *Portugal* was free from yellow fever down to 1850; in that and the following year isolated cases were observed at Oporto among custom-house officers and other persons who had been employed on board several vessels that had arrived with yellow fever. The fever showed itself again there in 1856 under the same circumstances, but to a greater extent: from 21st July to 2nd October 120 persons sickened and 53 of these died, and isolated cases occurred also at Belam and Lisbon. The epidemic at Lisbon was not until the following year, and it spread thence to Belam, Olivaes, and Almada.[3] Since that time there have been occasional importations (1858, 1860, 1864) of cases of yellow fever into Portuguese harbours, but they have not given rise to an epidemic outbreak.[4]

Ships with yellow fever on board have several times arrived at other ports in Western Europe, as, for example, Brest[5] in 1802, 1839, and 1856, St. Nazaire[6] in 1861, Swansea[7] in 1843, 1851, 1864, and 1865, and Southampton[8] in 1852, 1866, and 1867. However, severe precautionary measures, aided doubtless by the conditions of weather, have in most cases prevented the disease spreading from the ships and from the quarantine stations; and only in a few instances, as at Brest in 1856, St. Nazaire in 1861, and Swansea in 1864, has it been communicated to a number of persons, especially custom-house officers and ship labourers who had come into direct contact with the infected vessels; at St. Nazaire, also to the crews of certain craft anchored in the neighbourhood of the originally infected ship. But it has never got to be generally diffused among the inhabitants of the ports.

A glance at the distribution area of yellow fever as here sketched, shows that the whole west coast and by far the greater part of the interior of North America, and the whole

[1] 'Lancet,' 1870, Oct., p. 483.

[2] Account in the 'Lancet,' 1878, Nov., p. 641, and Guichet.

[3] See 'Relatorio da epidemia de febre amarilla em Lisboa no anno 1857.' Lisboa: 1859, Guyon (II), Lyons.

[4] Account in the 'Journ. de la Sociedad das Sc. Med. de Lisboa,' 1864.

[5] Keraudren (I), p. 19, Bertulus (I), Chervin (II), Beau.

[6] Melier.

[7] Buchanan.

[8] Wiblin, accounts in the 'Med. Times and Gaz.,' 1866, ii, 557, 590, 672, and in the 'Lancet,' 1866, ii, 550, 1867, i, 119, ii, 569.

of Africa and Europe, excepting the two regions named, have hitherto remained quite undisturbed by the disease. All that has been said about the occurrence of yellow fever in Europe before the discovery of America rests upon errors of diagnosis, and the same fallacy underlies the statements about the supposed prevalence of the disease in Asia Minor, India, the East Indies, and other insular or continental regions of Asia, which, like Australia, has *never* been visited hitherto by yellow fever.

The *geographical limits of the yellow fever area* extend, in the Western Hemisphere, to 34°54 south latitude (Monte Video), and 44°39 north (Halifax), in the Eastern Hemisphere to 8°48 S. (Ascension), and 51°37 N. (Swansea); but if we have regard only to the epidemic occurrence of the disease, then the parallel of 43°4 in the Western Hemisphere (Portsmouth, N. Hamp.), and that of 43°34 in the Eastern (Leghorn), form its northern limits.

§ 81. Characteristics of an Epidemic.

Yellow fever has occurred as a *pandemic* only on rare occasions. These are the later years of the eighteenth century (1796-98), the years 1819-20 (when the disease attained a considerable diffusion in Spain also), the year 1839, and then a quick succession of years, 1852 to 1853 (in which period the general outbreak on the South American continent also took place), 1855-56, 1866-68, 1873, and 1876-78. The increasing frequency and extent of these pandemic outbreaks have been obviously in proportion to the greater facilities of intercourse among nations, and especially to the development of the traffic by sea. On the other hand, as an *epidemic* prevailing to a limited extent at more or less numerous places, and often at merely scattered points, yellow fever is a permanent form of disease within the limits of distribution which we have already traced; so much so that the annals of pestilence, from the year 1791 onwards, can show very few years quite free from epidemics of yellow fever. Even the years exempt from epidemics may be taken

as only apparently exempt, inasmuch as the large gaps in the epidemiological records from many parts of the West Indies and Mexico, render it impossible to give a perfectly accurate statement of the diffusion of the malady in time and place.

An epidemic outbreak of yellow fever never happens suddenly; a series of isolated cases always precede it for a longer or shorter period (four to eight weeks or more), after which the epidemic will usually come rapidly to a head; it will exhibit many fluctuations while it lasts, depending chiefly upon the influx of strangers, and will die out gradually, or, it may be, suddenly, under the influence of conditions of the weather to be afterwards mentioned. As regards their range, the various epidemics show great differences. Often the disease will remain limited to one quarter of a town, which forms its chief if not its exclusive seat in all yellow fever epidemics that occur in the place; that is known to have been very decidedly the case in Charleston, Baltimore, Philadelphia, New York, and Boston, and it has been partly the case in New Orleans, and in many of the towns in Spain that have been attacked more than once. Next, the disease may spread through several quarters of a town, and sometimes even over the whole town; but, even under those circumstances, it is always at a few points that it concentrates itself—in single houses, or blocks of houses, or streets, not unfrequently sparing the houses close at hand. This tendency of yellow fever to form particular foci of infection, reminds us strongly of the mode of diffusion in the case of cholera; and that resemblance is manifested not less clearly in the fact that the disease is often confined exclusively to ships lying in the harbour or in the roadstead.

Just as in their area or range, so also in their duration, the epidemics of yellow fever exhibit great differences. In many cases, they last only a few weeks or two or three months; another time, the epidemic is protracted over half a year or more, or it survives even several years, during which period numerous fluctuations of intermission and exacerbation will occur, depending partly on conditions of the weather, partly on active movements of the population (influx of strangers, arrivals of troops, &c.). There are

hardly less marked differences in the frequency with which the several localities or regions are visited by the sickness, as we may see in the foregoing chorographic tables of the epidemics in the West Indies, the United States, Central America, and the West Coast of Africa.

§ 82. Influence of Race, Nationality, and Acclimatisation.

One of the most interesting points in the history of yellow fever comes up in considering the influence which *circumstances of race, nationality, and acclimatisation* exert upon the distribution of the disease.

New arrivals most exposed.—At those points of the yellow-fever zone where the disease bears the character of a more or less permanent sickness—whether endemic, or continually reimported, matters not—it is especially the newly-arrived strangers or those not yet acclimatised that are subject to the disease; while the natives, creoles, and acclimatised immigrants enjoy an exemption from it more or less complete.

On this point there is perfect unanimity among observers in all places and at all times; the behaviour of the disease towards the inhabitants of the yellow-fever zone cannot be expressed better than in the words of La Roche (ii, p. 25): "Within the tropics, the population consists of two classes:—the first composed of the natives and acclimatised, who, so far as relates to the fever in question, live with immunity amid the sick and the dying; the second of strangers, who are almost invariably attacked by the reigning disease and perish in a large proportion." It is this circumstance that explains the often observed fact, which has been already mentioned several times, that the disease, after subsisting for a time in isolated cases only, suddenly attains the proportions of an epidemic on the arrival of large bodies of strangers (numerous arrivals of ships, movements of troops, immigration on a large scale), committing terrible ravages among the new comers, while the native or acclimatised part of the population remain to the last almost exempt.[1]

Special liability of strangers from northern latitudes.—

[1] Instances of this are given by Leblond, p. 226, and Kerhuel for Cayenne, Lègris for Mexico, Lemprière, ii, 52, 55, for Jamaica, Desportes, i, 166, and Moreau (I), p. 74, for San Domingo, Davy, p. 281, for Barbadoes, Wurdeman for Charleston, Fortin, p. 312, and Drake, ii, 195, for New Orleans, and by Posey for Savannah.

The degree to which this liability of strangers rises, will depend to a certain extent upon their *nationality*, or, in other words, upon the temperature of their native country; for the predisposition increases in proportion as they come from higher latitudes, and that ratio obtains not only for the total amount of sickness, but also for the total mortality. "The mortality of the vomito to the new-comer from the cooler latitudes," says Townsend (II, 339), "may be said to be in an exact ratio to the distance from the equator of his place of nativity and residence."

Out of a large number of data supporting this fact,[1] I specially select the following. Blair states (p. 59), from experiences in Guiana from 1837 to 1845: "The lower the winter isobar in the native country of those attacked, the more severe was their sickness; so that, while the mortality among West Indians amounted to only 6·9 per cent. of the sick, it rose to 17·1 among the Italians and French, 19·3 among the English, 20·2 among the Germans and Dutch, and 27·7 among Scandinavians and Russians." According to the account of Barton, there occurred, per thousand deaths in the New Orleans epidemic of 1853:

Native Creoles	. . . .	3·58
Strangers from	West Indies, Mexico, and S. America . . .	6·14
„	„ Southern States of the Union .	13·22
„	„ Spain and Italy . .	22·06
„	„ Middle States of the Union .	30·69
„	„ New York and New England States . . .	32·83
„	„ Western States of the Union .	44·23
„	„ France . . .	48·13
„	„ British America . .	50·24
„	„ Great Britain . .	52·19
„	„ Germany . . .	132·01
„	„ Scandinavia . . .	163·26
„	„ Austria and Switzerland .	220·08
„	„ Netherlands . . .	328·94

Acclimatisation.—The *immunity from yellow fever conferred by acclimatisation*, which is moreover not an absolute one, is only got by a residence of many years in a locality constantly visited by the disease, or, still more surely, by having

[1] See Chisholm (II), 144, Taylor, p. 205, Savarésy, p. 260, Bally (I), pp. 268, 334, Dickinson (II), p. 13, Dickson (II), p. 257, Arejula (II), p. 325, McKinlay, p. 340, Arnold, p. 26, Zimpel, p. 68, Lallemant, p. 21, Wucherer (I), Jewell (I), Bernard, p. 21.

come safely through one attack; a residence—even a prolonged residence—in a place which is situated within the yellow-fever zone but is seldom or never troubled by that disease, diminishes the individual predisposition only a little, and, as it seems, really to no greater extent than residence in tropical or subtropical regions in general.

"The chances of immunity," says Dutroulau (IV), p. 369, "appear to be always in direct proportion to the length of residence at the head-quarters of the disease; but no acclimatisation is acquired except by those who have lived through a previous epidemic period without quitting the country, and who have been more or less impregnated by the epidemic principle, and, most of all, by those who have survived an attack of yellow fever." To the same effect, Dowler says (I), p. 37: "It is the resident city creole, not the country creole—not the creole who migrates every summer to New York, London, or Paris—that may hope for as good health as is possible to humanity, while two or three hundred others daily fall victims around him." From observations made in Charleston, Simons draws the conclusion: "All persons who have not spent a yellow-fever year there are liable to the disease, and it is questionable if they are wholly exempt until they have had the disease;" and Rufz (p. 628) informs us of the remarkable fact that, on the island of Martinique, which quite escaped the yellow fever from 1826 to 1838, numerous individuals sickened (for the most part slightly) in the epidemic of the latter year, although they had spent six to ten years on the island. In every hundred deaths from yellow fever among the strangers in Rio Janeiro in 1876, the length of residence, according to the data of Rey (p. 382), had been as follows:[1]

41	had resided	in	Rio	from 1 day to 6 months.
39	„	„	„	6 months to 1 year.
14	„	„	„	1 year to 2 years.
4	„	„	„	2 years to 3 years.
2	„	„	„	4 years to 6 years.

There are numerous statements to show that individuals who had been born in the immediate neighbourhood of the yellow fever foci and lived for a long time there, have taken the disease as soon as they exposed themselves to the endemic influences outside their home; such are the statements of Ramsay (IV) and Dickson (II), p. 257, for Charleston, Dowler, p. 35, for New Orleans, Kerandren, p. 23, for Martinique, Imray for Dominica, Fergusson (IV), p. 181, for Barbadoes, Crouillebois, p. 434, Moufflet, Heinemann (I), p. 164, (II) p. 159, and Watson for Mexico.

[1] Further information on this subject will be found in Keraudren (I), p. 24, Imray (I), p. 94, Fergusson (IV), p. 144, Doughty, p. 64, Heinemann (I), p. 164.

These facts enable us to understand, in the next place, how it happens that an individual rarely sickens with yellow fever more than once. Some observers even consider a second attack to be out of the question;[1] while others go so far as to admit that a second *severe* attack occurs only in those who had the disease at first very mildly or, contrariwise, that there may be a slight second attack when the first had run its course with severe symptoms; and that, for the rest, a recurrence of the sickness occurs for the most part only under certain circumstances, which, as we shall see, override altogether the immunity gained by acclimatisation, or render it at least relatively insufficient. It follows, then, from the facts just mentioned, that there can be no question of acclimatisation at all, for regions which do not form permanent foci of yellow fever; for regions where the disease occurs so rarely that the various outbreaks are separated from one another by an interval of many years, in which the natives and the old-established settlers sicken in large numbers and often mortally, and are better off than the strangers only in so far as the circumstances of climate in which they have lived, make them less predisposed to the illness than the latter.[2]

It appears to be still doubtful whether there is any *congenital immunity from yellow fever due to peculiarities of race.* So far as relates to all the nationalities of the *white race,* the idea may be set aside altogether; while the relative immunity that the creoles enjoy is partly referable to acclimatisation, partly to the circumstance that many of them had already got over the disease in childhood and so become immune.

"Those native children," says Shecut (p. 108), "that arrive at the age of nine years, are thenceforward considered as naturalised to the climate; but until then, they stand equally exposed to the disease with

[1] Seaman, p. 40, Lining, Dickson (II), p. 273, Archer, p. 61, Nicholson, p. 810, Barrington, p. 311, Strobel, p. 202, Dowler, p. 35, Arejula (II), p. 290.

[2] There can be no doubt that the cases of repeated attacks of yellow fever are much rarer even than the observers admit, as it can be shown that many errors of diagnosis have occurred (confusion between yellow fever and severe bilious remittent malarial fevers) which have made a decision difficult on this as on many other points.

strangers or foreigners;" and Lota ('Arch. de méd. nav.,' 1870, Oct., Dec., xiv, p. 344) states: "The fevers that attack the creole children during epidemics of yellow fever are more or less pronounced forms of that disease, and the immunity that the adult creoles enjoy with respect to yellow fever, provided they have not left their country in infancy, is not an advantage that they owe to race or climate, but their preservation is due to their having had the disease before." Heinemann (I, 164) takes the same view, on the ground of experiences that came under the notice of himself and other careful observers at Vera Cruz. The statement that children are exempt from yellow fever is shown to be entirely groundless by the observations made in Antigua by Chisholm (II, p. 281), in Guiana by Blair (II), at Charleston by Dickson (p. 257) and Simons (I), in Martinique by Rufz (p. 127), at New Orleans by Gros and Gerardin (p. 7), and at Curaçao by the Dutch physicians ('Nederl. Tijdschr voor Geneesk.,' 1860, p. 256); and when Faget (p. 32), Mercier, St. Vel, and others express the opinion that those illnesses in children which are designated as yellow fever are nothing else than severe cases of malarial fever, it might be retorted with better reason, as Heinemann has already pointed out, that the alleged exemption of children from yellow fever depends on the fact that the cases of sickness occurring among them are always mistaken for malarial fevers.

Immunity of the pure-blooded negro.—For the *American Indians* (redskins), according to experiences in North America, Mexico and Brazil, and for *coolies* (Hindoos), according to the experience of Kerhuel in Cayenne, there can be just as little thought of congenital immunity from yellow fever as in the case of the white race. On the other hand, such an immunity cannot well be considered as out of the question for the *black or negro race.* The same may be true also of the *Mongolian stock;* at all events, Eysaguirre states (p. 12): "The Chinese settled at Lima are, like the native negroes of the country, almost exempt from yellow fever."

"It is a well-established fact," says Fenner (p. 56), "that there is something in the negro constitution which affords him protection against the worst effects of yellow fever, but what it is I am unable to say." Doughty (p. 30) expresses himself to the same effect: "In the natives of Africa the constitution appeared to me as secure against yellow fever, as a person who has had the smallpox is against its recurrence." Many other experienced physicians[1] say that they had never seen a case

[1] Arnold, p. 34, Moultrie, p. 4, Leblond, p. 161, Curtis, p. 244, Dickson, 'Essays,' i, 345, Hume (I), p. 230, Bally (I), p. 305, Eysaguirre, Gouin, Hille, p. 38, Crouillebois, p. 434, Schmidtlein, p. 56, La Roche, ii, p. 65.

of yellow fever in the negro, while others[1] cannot but admit that the disease occurs much more rarely in negroes, and runs a much milder course, than in other races.

That we have here to do with a congenital and not an acquired immunity in the negro race may perhaps be inferred from the circumstance that the same immunity occurs in a remarkable way among negroes who have not been acclimatised in the yellow-fever zone, and that the immunity is all the more complete the more purely the racial characteristics of the individual have been preserved.

The former of these assertions is confirmed by Daniell, who mentions (p. 64) that in the epidemic of 1820 at Savannah, not a single case of sickness from yellow fever was made out among 300 negroes that had just been imported; it is confirmed also by Blair, who remarks of the epidemic in Guiana in 1852-53 that: "Of 7890 African (black) immigrants, none contracted yellow fever;" and further by Reynaud and Bouffier, who agree in pointing out that among the 500 negroes from the Soudan and Nubia—regions free from yellow fever—who accompanied the French army to Mexico, and were all the time in the fever zone there, not a single case of yellow fever occurred, whereas the French and Mexicans were decimated by the sickness. Respecting the second point, Nott states: "I have seen the disease prevail five times at Mobile and have attended several hundred cases among the whites, but not a single well-marked one in a pure-blooded negro, and not more than two or three in mulattoes." To the like effect Fenner remarks: "The least mixture of the white race with the black seems to increase the liability of the latter to the dangers of yellow fever, and the danger is in proportion to the amount of white blood in the mixture." The same view is taken by Bryant (p. 299), in conformity with observations made by him in the epidemic of 1855 at Norfolk (Va.); and Clarke (for the Gold Coast), Tidyman (p. 325), Nicholson (p. 856), Lewis (III) (p. 416), and others agree with him in saying that yellow fever is much commoner among mulattoes, quadroons, and other varieties than among pure negroes.

While it is necessary to admit, according to this evidence, a more or less complete immunity from yellow fever, either congenital or acquired, under the circumstances stated, it is, on the other hand, certain that this immunity may be lost

[1] Rufz (II), p. 127, Dowler, p. 38, Tidyman, p. 325, McKinlay, p. 340, Sarrouille, p. 36, Wucherer (II), p. 393, Mallory. In the epidemic at Shreveport in 1873, the mortality among the white population was nearly 26 per cent., while only 6 per cent. of the coloured population died (Jones (III), p. 151); the difference was still greater in the epidemic of 1876 at Savannah, where, according to Woodhull, the respective mortalities were as 15 to 2.

again under other circumstances, or may prove insufficient. As regards the first point, the most important consideration is a change of residence, or a considerable time spent by the acclimatised person outside the yellow-fever zone, and especially in higher and colder latitudes; so far as concerns insufficiency, that depends on the severity of the epidemic.

"Like West Indians," says La Roche (ii, 31), "the acclimatised inhabitants of our southern cities lose the protection they possessed, by a prolonged residence in some northern place or in a rural district in the same region." Heinmann's opinion, after many years' observation in Vera Cruz, is: "Even foreigners may remain insusceptible to the disease for a considerable number of years, provided they do not leave the focus of disease during that period. An absence of a few months only is sufficient to take away this immunity. . . . That the congenital safeguard against yellow fever becomes lost even in natives by having been away for years is a fact long recognised; I mention the two following cases merely because they made an unusual sensation in the place. A colonel in the Imperial service, a native of Vera Cruz, found it necessary, after the overthrow of the Empire, to go to Europe, where he lived three years; after his return he sickened so severely with the vomito that his recovery was for a time doubtful. A second Vera Cruzan, well advanced in years, who had been at the head of the administration of the military hospital for thirty or forty years, died there in the course of last year's (1878) epidemic, having spent the few years preceding as a resident in the capital (City of Mexico)." By far the larger number of observers express themselves to the same effect.[1]

Benefit of acclimatisation may be lost.—According to the assertions of several authorities, the immunity got by acclimatisation proves effective only at the place where it was acquired; so that it vanishes with a change of residence, even when that does not involve conditions essentially worse than those left behind.

Humboldt (p. 338) has already drawn attention to the fact that the natives of Vera Cruz may enjoy an absolute immunity from yellow fever at home, but when they emigrate to Havana or to one of the pestilential centres in the Southern States of the Union they die of the disease there sooner or later. Facts of the same kind are

[1] Wucherer is led by his own observations, and on the authority of Pym, to give a decided opposition to this opinion; he considers an immunity once acquired by acclimatisation to be an absolute one. Without questioning the accuracy of the observations made by that author, I do not consider them conclusive as against the great majority of other observers, and the testimony of Pym is of still less account.

given by Doughty (pp. 58, 65), Pugnet (p. 346), and others. This helps to explain the danger involved in removing European troops from one island of the West Indies to another. Let two bodies of troops, says Cornuel ('Annal. marit.,' 1844, ii, 739), stationed at different points in the Antilles, and living under the most satisfactory conditions of health, exchange garrisons, taking every precaution, and cases of yellow fever will shortly break out at both places without there having been the slightest change in the conditions of the locality; while the previous state of good health will continue undisturbed among those of the troops who were left in the garrisons. As regards the second point (immunity lost owing to the severity of the epidemic), it is worthy of note that it was precisely the severe epidemics of yellow fever, such as that of 1876-78 at Vera Cruz, those of 1796, 1799, 1819, 1833, 1847, and 1853 at New Orleans, those of 1817 and 1849 at Charleston, 1821 at Wilmington, 1805 and 1807 in Jamaica, 1817 at Trinidad, and 1852 in Martinique, in which cases of sickness and death were frequent (and in some of them excessive) among the creoles and acclimatised strangers.

What has here been said as to the loss or weakening of acquired immunity appears to hold good in an equal degree for the congenital immunity of the negro race.

"Africans who have travelled to Europe or to higher latitudes in America," says Jackson (p. 146), "are by no means exempt from the disease when they return to the West Indies;" Lemprière (ii, 29) adheres to this statement, adding: "This remark has been fully confirmed by my own experience during the present year (1792-93)," and similar observations have been published by Veitch,[1] Bancroft (p. 274), and others. It is further noteworthy that, in the opinion of thoroughly reliable observers,[2] negroes sicken in Senegambia, on Boa Vista, the Benin and Biafra coasts, Teneriffe, the parts of North America that are seldom visited by yellow fever, as well as in Guiana and Brazil, more often and more severely than on the coast of Sierra Leone, in the West Indies, on the Gulf Coast, or at places in general where yellow fever is constantly prevalent. Lastly, it holds good for the negro race also, that their peculiar immunity proves insufficient in severe epidemics of yellow fever. Among numerous examples of this may be mentioned those furnished by the New Orleans epidemics of 1819, 1820, 1822, 1833, and 1853,[3] that of 1878 at many places in the Southern States of the Union,[4] those of 1799, 1807, 1824, and 1827 at Charleston,[5] the epidemic

[1] 'Letter to the Commission for Transports of Sick and Wounded Seaman, &c.,' London, 1818, p. 112.

[2] La Roche (II), p. 64, Dutroulau (IV), p. 369, Vergoara, McWilliam, Thevenot, p. 254, Cedont, p. 344, Rush (I), p. 117, Seaman (I), p. 6.

[3] Valentin (II), p. 89, Dupuy, Thomas (I), Barton (I), p. 47, Fenner (I), p. 54.

[4] McMeurtry.

[5] Valentin, l. c., Ramsay (VIII), Simons (I), Dickson (IV).

at Mobile in 1819,[1] at Norfolk in 1855,[2] at Pensacola[3] in 1822, in Guiana[4] in 1837 and 1850, in Guadeloupe in 1795,[5] in Antigua in 1835,[6] and in Sierra Leone in 1823.[7]

§ 83. A Disease of the Tropics and the Hot Season.

A glance at the zone of yellow fever—that region of the globe where yellow fever is always prevalent, or at least often occurs as an epidemic, whose range extends in the Western Hemisphere between 32°46 N. (Charleston) and 22°54 S. (Rio de Janiero), and in the Eastern Hemisphere between 14°53 N. (Cape Verd) and 5°7 N. (Cape Coast Castle) —shows that the disease bears the marked impress of a *tropical malady*. It is not less a tropical malady, as the sequel will show, because some regions of the tropics which lie within the yellow-fever zone are exempt from it, or because they are visited by it only exceptionally and under the same circumstances as places lying outside the zone, for they enjoy a temperate climate in consequence of their elevation. The pathogenesis appears, therefore, to be essentially linked with *tropical climate*, and that assumption is completely borne out by the fact that the disease is found to be dependent for its origin upon the season and the weather, and more particularly upon the temperature.

Within the tropical part of the yellow-fever zone, the West Indies and those parts of the Mexican and West African coasts that are visited by the disease, yellow fever occurs in sporadic and endemic form *at all seasons*;[8] but the greatest prevalence of the disease falls in the period from April to September.

[1] Drake.
[2] Williman, p. 165.
[3] Drake.
[4] Kerhuel, Fraser.
[5] Bishopp in Pym, p. 118.
[6] Furlong (I), p. 290.
[7] Boyle, p. 270.
[8] Williams (I), p. 3, Belcher, p. 248, Savarésy, p. 32, Miller (III), p. 137, Bally (I), p. 309, Valentin (II), p. 88, Hillary, p. 175, Bouffier, p. 529, Heinemann (I), p. 165, Boyle, p. 204.

According to the data of Bouffier (p. 529) there were received into the Marine Hospital of Vera Cruz 6941 cases in the course of thirty-two years, the monthly admissions having been as follows:

January	151	July	815
February	210	August	769
March	437	September	720
April	683	October	494
May	1058	November	281
June	1078	December	245

According to this the number of patients admitted from April to September was 5123, or 73·8 per cent. of the whole. In 111 epidemics of yellow fever in the West Indies and on the coast of Mexico, of which the time of prevalence is more exactly given, the beginnings and endings were as follows:

	Began.	Ended.		Began.	Ended.
January	2	7	July	9	8
February	5	6	August	8	8
March	7	1	September	15	8
April	16	1	October	8	8
May	9	2	November	4	13
June	22	3	December	6	5

On the Sierra Leone coast all the epidemics except one (that of February, 1823) commenced between April and June; on the other hand, for reasons to be afterwards given, they commenced on the Senegambia coast between June and October. In the regions of South America situated within the northern tropic, and belonging to the yellow-fever zone, the epidemic prevalence of yellow fever falls chiefly in the months from August to December; although some epidemics (in Surinam 1835, Demerara 1851) have not begun until December, and others (in Berbice 1803, British Guiana 1852, Cayenne 1856) not until January. Lastly, in Brazil and Peru the yellow-fever season has hitherto been the summer and autumn (January to June).

Of 8554 fatal cases of yellow fever at Rio de Janiero during the period from January, 1851, to July, 1870, there occurred in the respective months:

January	1118	7597 = 89 %	July	242	957 = 11 %
February	1760		August	164	
March	1732		September	108	
April	1434		October	104	
May	996		November	116	
June	557		December	223	

It appears, then, that the time for epidemics of yellow fever is chiefly, but not by any means exclusively, the hot season and the rainy season in those latitudes of which we have been speaking. The connexion between the disease and particular seasons of the year comes out still more prominently in its epidemic prevalence in countries situated in

higher latitudes. The following table gives a conspectus of those yellow-fever epidemics of the United States and Bermuda, for which the time and prevalence has been more exactly recorded.

	New Orleans.		Texas and Louisiana.[1]		Miss., Alab., Georg., Flor., Tenn.		Charleston.		N. Car., Virg., Maryland, Delaware.		Philadelphia and New York.		N. Eng. States and New Jersey.		Bermuda.		Total.	
	Began	Ended															Began	Ended
January . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
February . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
March . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
April . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
May . . .	2	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	3	...
June . . .	3	...	...	...	4	...	3	...	2	...	4	...	2	...	1	...	19	...
July . . .	15	...	...	...	10	...	9	...	6	...	17	...	4	...	2	...	63	...
August . . .	2	...	4	...	16	2	11	...	8	...	7	1	9	1	3	...	60	4
September . . .	2	...	13	...	10	3	2	4	1	2	1	2	2	2	...	...	31	13
October . . .	...	8	2	2	2	3	...	10	...	7	...	12	...	5	...	2	4	49
November . .	...	4	...	3	...	12	...	4	...	4	...	7	...	3	...	2	...	38
December . .	...	2	...	...	...	4	...	...	...	...	...	2	...	1	...	2	...	11

The following conclusions may be drawn from this table. During the first four months of the year, yellow fever has *never* been epidemic in the countries in question; only three epidemics began in May, and these occurred in two towns (New Orleans and Charleston) which have both been visited by yellow fever unusually often; the number of epidemics occurring in June (19) is comparatively small; July and August are the months in which by far the most of the epidemics have begun (123 out of 180 epidemics); next to them comes September (31), while in October the disease began only four times (again in the most southern or Gulf Coast States), and in November and December it did not break out once. The close of the epidemic falls mostly in the months of October and November (87 times in 115 epidemics); only 11 epidemics have lasted into December, and none have gone beyond that month. Summer and autumn therefore are the proper yellow-fever seasons of those regions; the epidemics occurring before June or after September concern the States lying farthest to the south. Thus

[1] Excluding New Orleans.

the principle formulated by Barton (p. 285), that the extension of yellow fever from the tropics towards higher latitudes is regulated by the rising temperature, and that the disease appears later in the respective regions the farther north these are situated, is a law that has only a limited application. On the Iberian peninsula yellow fever has always occurred during the period from July to September; some localities it has not reached in the course of its progress before October, and it has died out always and everywhere in December at the latest.

§ 84. Relations to Heat and Cold.

It will appear that this relation of epidemic yellow fever to the season is determined by the kind of weather, depending above all upon the range of the *temperature*. Yellow fever, as a permanent disease, lasting, that is to say, the whole year through, and becoming sometimes epidemic even during the cold season, is found only in those regions where the mean winter temperature amounts to at least 20—22° C. (68—72° Fahr.), as on the Sierra Leone coast, in the West Indies, the northern coast belts of South America, and the Mexican coast; and in these the malady does not usually attain to epidemic diffusion until the hot season, and seldom before the temperature has reached a height of 26° C.[1] (79° Fahr.). In higher latitudes, with an isotherm of less than 20° C. or 68° Fahr. (New Orleans, Mobile, Key West, Charleston), yellow fever occurs as an epidemic only in years when the temperature comes up to that of tropical regions; and then, also, it is principally in the hot season that it occurs. Lastly, in places with a still cooler climate (Central States of the Union, New England States, parts of Europe where the fever has been) the disease occurs almost without exception in the hot season only; it has never grown into an epidemic except when the heat has equalled the mean annual temperature of the tropics, *and it has on no occasion become diffused in a temperature below* 20° *C.* (the winter temperature of the tropics). The same influence of a high tempera-

[1] Walter, in Bernard, p. 20, Crouillebois, p. 430, Humboldt, Hillary, p. 175, Valentin (II), p. 88, Belot, and various others.

ture upon the pathogenesis is shown also in the fact that an increase of the epidemic takes place not unfrequently in proportion to the rising heat, and above all in the fact that a considerable fall of the thermometer has always been followed by a considerable remission, while a *frost* has brought the epidemic to an end under all circumstances whatsoever.

"During the ten years," says Dalmas (p. 19), "that I have spent on the continent of America, I have always seen yellow fever following great heats and ceasing with the first cold." As to the extinction of the disease under the circumstances of a low thermometer, we find reliable observations from higher latitudes—for the New Orleans epidemics in 1819-20-22, and -33, Opelousas 1837, Galveston, Franklin, and Alexandria 1839, Natchez 1817-23, and 25, Vicksburg 1841, Mobile 1819 and -54, Key West 1867, Charleston 1745-48, and -52, Memphis 1873, Norfolk 1795 and -97, Baltimore 1800-8, and -19, Wilmington 1802, Philadelphia 1699, 1741-47-62-97-98-99, 1802-05, and -20, New York 1791-98, 1805, and -22, Malaga 1741, Gibraltar 1804, Barcelona 1821 and 1870. The influence of this factor is manifested also within the tropics (Brazil, Peru, West Indies, Vera Cruz) in the usual remission or cessation of the epidemic on the setting in of the cold weather.

At the same time it has to be noted that a yellow fever epidemic is not dependent for its continuance upon a certain temperature (minimum of 20-22° C.) in the same way that it is dependent for its development; an epidemic once developed may continue with a low thermometer, and a complete extinction of the pestilence can only be counted on *when the temperature falls to freezing point.*

Drake (II, p. 194) concludes from his investigations: "That a heat of 80° F. or upwards is necessary *to the rise of the fever*, but that having become prevalent it will continue under a lower temperature than that which is necessary to its production." Particular proof of this is furnished by the following epidemics:—Lower Louisiana 1853 (*Dowler*), Mobile 1843, Charlestown 1849-52 and -54, Memphis and Shreveport 1873, Norfolk 1800 and 1801, Philadelphia 1800, New York 1795, Malaga 1803 (the sickness continuing, as Keraudren tells us, after the neighbouring mountains were covered with snow), Gibraltar 1813 and -28, Lisbon 1857, Newcastle (Jamaica) 1866-67. Townsend also concludes from his observations made in New York from 1798 to 1822, that the continuance of a yellow-fever epidemic will certainly be helped by a high temperature, but is not absolutely dependent upon it, and that the sickness is as likely as not to last under a low temperature so long as the mercury does not touch freezing point. And Fearn (in Drake, II, 224) formulates from his experiences in Mobile the rule that: "The cold, which merely produces white frost, will not finally check the dis-

ease (the temperature of the ground need not fall below 40° F., for this effect to be produced); *to terminate an epidemic, ice must form on the surface of the ground.*"

There are a good many facts to warrant the inference that this extinction of the epidemic on the setting in of frost does not carry with it a complete destruction of the morbid poison. In those regions where the poison can be shown to have been imported, more especially in Spain, it has on several occasions survived the winter and exerted its powers anew, or given rise to a fresh epidemic outbreak, when the temperature has risen again next year; as in Cadiz and Medina Sedonia in 1800-1801, Malaga 1803-1804, Murcia 1811-1812. Or, it may be that the epidemic, having been checked by frost, has revived when the warm weather has returned later in the season; as in the examples of 1801 in Block Island (*Willey*, p. 103), Jumilla 1811 (*Velasquez*), Barbadoes 1816-17 (*Ralph*), New Orleans 1837 (*Thomas*, p. 60), and Memphis 1879. The observations made on board infected ships during a voyage through various latitudes are especially instructive in this respect.

Keraudren (p. 18) mentions several cases in which yellow fever had developed on board ships on the Gulf Coast, had persisted during the voyage into temperate latitudes, although the heat was much below 19° C., had died out at last, as often happened with ships going from the West Indies to the coast of Newfoundland, but had broken out anew as soon as the ships sailed into southern latitudes after a somewhat short stay in northern waters. Barrington (p. 309) publishes the following observation: the infected ship-of-war *Hornet* arrived on the 29th of October, 1829, at the harbour of Pensacola; the temperature there having fallen to 20° C. (68° Fahr.), only two new cases of sickness occurred, and when the ship left that port for New York, the disease appeared to have been completely extinguished. But when they were off the south cape of Florida, in a temperature of 25°—28° (77°—83° Fahr.), numerous fresh cases of illness from yellow fever occurred among the crew, and the sickness did not completely die out until the vessel reached higher latitudes and the thermometer had gradually fallen to freezing point.[1] The effect of cold upon the extinction of the sickness was manifested with remarkable clearness on board the ship *Narva* which was employed in laying the telegraph cable between Cuba and Florida in 1867. On the 17th and 18th of September, according to Dunlop's account (p. 211), fresh cases of yellow fever were still occurring as the vessel lay at

[1] See also Maccoun's narrative relating to the *Susquehanna* in the year 1858, and the report of the Surgeon-General of the United States Navy in the 'Philad. Med. and Surg. Reporter,' 1879, April, p. 351.

anchor in Key West; when the ship left the harbour on the 21st, the thermometer suddenly fell 15° F., and the disease was put an end to as if at a single blow. Not always, however, have matters gone so favorably. It has repeatedly happened that infected ships have arrived at North American and European ports late in the year and in very cool weather with yellow fever on board; and not only so, but new cases of sickness have in several instances occurred among the ship's company after arrival, as, for example, at Brest in 1839 and 1856, Halifax (N. S.) in 1861, and Southampton in 1866. There can be no doubt, as we have already indicated (p. 337), that nothing but the lateness of the season and the low temperature, has prevented the infection from extending from the ship to the population of the port.

§ 85. Influence of Moisture and Winds.

The amount of moisture in the air and the *precipitations from the atmosphere*, constitute a second factor in the production of yellow fever, but a less important one than the temperature. Some observers would make an atmosphere saturated with watery vapour almost up to the dew point, an essential condition for an epidemic of yellow fever. Thus, Barton, among others, states in his report on the New Orleans epidemic of 1853: "The epidemic yellow fever has never occurred here at its commencement but during a high dew-point, the minimum being upwards of 74°. . . . . Yellow fever has always ceased as an epidemic before the dew-point descended as low as 58°." This assertion finds support in the evidence adduced by La Roche (ii, p. 130) that all countries situated outside the tropics, in which yellow fever is a standing disease or now and then epidemic, have a high degree of atmospheric moisture during the hot or yellow-fever season. It is further noteworthy that the disease occurs only to a slight extent, or not at all, in years with little rain, or, it may be, in an unusually late season when the rains have been delayed; and that is the case not only in the tropics where the disease always develops on the setting in of the rains or directly after their cessation, but also in the extra-tropical regions of the yellow-fever zone. In the latter, the disease has been mostly prevalent in rainy weather,[1] or has become epidemic directly after the rains;

[1] See Dickson (II), p. 265, Porter in the 'Amer. Journ. of Med. Sc.,' 1854, Oct., p. 353, Hosack (I), 305, Townsend.

dry weather, on the other hand, and especially long-continued drought, has proved less favorable to the epidemic diffusion of the disease. Finally, very abundant and continuous rains in tropical as well as in extra-tropical regions, have not unfrequently brought the epidemic to an end.[1] The importance of this factor for the development of an epidemic ought perhaps to be ascribed to the modifying influences which atmospheric moisture and precipitation exert upon the temperature of the air; perhaps, also, to the dependence upon the same of the decomposition-processes in organic matters, or the growth of lower organisms connected therewith. But we are the less able to come to a definite opinion on this point, inasmuch as there are a good many observations that go counter to the facts above adduced; as, for example, that in certain tropical regions belonging to the yellow-fever zone, such as the coast of Guiana[2] and the Basse-Terre of Guadeloupe,[3] dry weather is especially favorable to the spread of the disease.

Influence of winds.—The conjecture that the remission or even complete extinction of an epidemic after abundant and continuous rain has its ultimate cause in the lowering of temperature thereby caused, obtains some support from observations made on the modifying influence of *currents of air* or *the wind*, upon the behaviour of the sickness. On the Mexican coast and in the West Indies, it is the winds from the south—in the one case south-east[4] and in the other south-west[5]—that materially help to induce the disease through raising the temperature, while cold winds from the north and north-east have often had a very good effect upon the state of health by moderating the heat.[6] On the Gulf Coast and the Atlantic coast of North America it is likewise the winds from the south—south or south-east according to

[1] See Leblond, p. 197, Valentin (II), p. 88, McArthur in Dickson (I), p. 47, Arnold, p. 31, Lemprière (I), 26, Townsend (I), Gillespie (I), p. 137.

[2] Fermin, pp. 3, 18, Chisholm II, 196, Hille, p. 37.

[3] Cornuel in the 'Annal. Maritim.,' II, p. 735.

[4] Humboldt, p. 765.

[5] Desportes (I), 19, Bally, p. 361, Lemprière (I), 17, Ralph, Rufz (II), p. 129, Savarésy, p. 189, Lefort, p. 9, Arnold, p. 26, St. Vel, 'Traité des maladies des regions intertropicales,' Paris, 1868, p. 76.

[6] Leblond, p. 179, Valentin (II), p. 88, Schmidtlein, p. 52, Crouillebois, p. 430.

the geographical position of the place—that are most to be feared in this respect,[1] while cold winds from the north have brought about a remission or even an extinction of the epidemics in those regions also.[2] In like manner, winds from the south-east have fomented the sickness in Brazil (as at Rio de Janeiro in 1854); on the other hand, the setting in of the pamperos,—cold, tornado-like, south-west winds blowing from the Andes across the pampas,—was directly followed by its extinction in Buenos Ayres in 1858.[3] It may be inferred that this modifying influence of the wind upon the morbid conditions does not reside in any peculiar qualities that it possesses, from the fact that even the most violent storms, if they last only a short time and have no permanent effect on the temperature, leave the state of the epidemic quite unaffected. In evidence of this there are observations from Sta. Lucia and Philadelphia in 1802, Antigua in 1816, and Norfolk (Va.) in 1821. Just as little are we warranted by the facts before us in speaking of a "purifying" influence of thunder storms, or of any relation whatever between the *electrical states of the atmosphere* and the development, continuance, or extinction of yellow-fever epidemics.[4] How far the wind may be considered an active agent in carrying the yellow-fever poison, will be discussed afterwards.

§ 86. Seldom leaves the Sea-Coast or the Banks of Navigable Rivers.

Not one of all the infective diseases is so decidedly local in character, or seems to have its existence so much bound up with particular *circumstances of place*, as yellow fever; and if, for the present, we can form only a very imperfect

[1] Barton (I), 242, Waring, Kelly, p. 386, La Roche ii, 179. On the Spanish coast also, as Fellowes (p. 14) mentions, the warm Levanter blowing from the east has had a very unfavorable effect on the course of yellow-fever epidemics.

[2] As at Norfolk (Va) in 1795, Boston in 1798, New Orleans in 1822 and 1837, Baltimore in 1819, Natchez in 1823, and Charleston in 1849.

[3] Scrivener (I).

[4] Sheeut made experiments with an electrical machine upon the electrical tension of the air in the yellow-fever epidemic of 1827 at Charleston. The conclusions drawn from them are wanting in scientific value, just as the experi-

estimate of the relation between these various circumstances and the development and spread of the disease, yet their great significance for the pathogenesis cannot really be doubted.

Among the peculiarities of distribution which show the dependence of yellow fever upon locality, the first to arrest the attention is the association of the disease, not perhaps exclusively, yet to a very great extent, with *sea-coasts and the shores of great navigable rivers.*

"There is a primary fact," says Bernard (p. 18), "that ought to strike us, if only from its constancy, viz. that it is always in the territories washed by the sea, and never in the interior of a country that we can detect the presence of this scourge;" and if that declaration goes somewhat too far, yet it expresses the general conviction that has been forced upon all observers. This local limitation of the foci of sickness, which Drake (ii, 188), Zimpel, Faget (p. 68), Hume (VII, p. 145), and others have already called attention to, is most marked on the continent of North America. Among all the epidemics that have raged on that great region of the globe up to the year 1873, only two (Washington 1825, and Woodville, Miss., 1844) have occurred at places remote from great river-basins; and even these were only a few miles distant (nine to twelve English). It was only in the great epidemics of 1873 and 1878 that the disease penetrated far into the interior of several of the Gulf Coast States, following other routes than the course of the great rivers. The diffusion of the disease follows the same laws in the West Indies, according to Chisholm (II, p. 281, 288), Lind (p. 185), Moreau (I, p. 157), Bertulus, Dutroulau, and others; in Guiana according to Blair; according to Baker, Döllinger, Lallemant, and others in Brazil (where the sickness has travelled as far as the confluence of the Rio Negro with the Amazon, or nearly 1000 kilometres—620 miles—into the interior, but always along the shores of that broad river); and according to Mellico, Celle (p. 83), Goupilleau, and others in Mexico (where it has visited inland places, but only exceptionally (Heinemann), following the highways of traffic to Cordova, a distance of 100 kilometres or 62 miles). Again, on the West Coast of Africa, epidemic yellow fever has always been confined to points on the coast; isolated cases, imported from the coast, have indeed been observed in Dabon (Gold Coast) and Bakel (Senegambia), but, as Sarrouille explains (p. 36), the disease has never grown to be epidemic at

ments themselves were ill-judged. In like manner, no weight attaches to the conclusions upon the influence of atmospheric electricity as a disease-factor which Belot, Rufz, Bertulus, and others have drawn from the fact of an increase in the number of yellow-fever cases at the time of thunderstorms. Those who are fond of speculations of that sort will find a summary of the data relating to them in La Roche ii, 113 ff.

those places. We find a more considerable exception to the rule in the behaviour of yellow fever in the great epidemics on Spanish soil during the first twenty or thirty years of this century; on these occasions the disease not only penetrated far into the interior of the country, but it even appeared in epidemic form at many places remote from the larger rivers.

§ 87. Limited Altitudinal Range.

The influence of circumstances of locality upon the distribution of yellow fever is shown, in the second place, by the fact that the disease rages almost exclusively on the *plains*, and does not tend to spread at *a considerable elevation*, except in rare instances of unusually severe epidemics.

On the continent of North America we find the limit of altitude of the disease to be from 100 to 200 metres, or 350 to 700 feet (Memphis, Holly Springs, and other places in the Mississippi valley).[1] In Cuba the hilly country in the interior has completely escaped hitherto (Belot), and in San Domingo, Tobago, Sta. Lucia, Dominica, and other of the smaller Antilles, the disease has rarely been seen at a height above 200 metres (700 feet). Two elevated points form an exception to this, the one being Camp Jacob in Guadeloupe,[2] at a height of 550 metres (1800 feet), and the other Newcastle in Jamaica, at a height of 1200 metres (4000 feet),[3] the highest point to which yellow fever has ever yet attained; but at each of the places the disease has been epidemic only once or twice altogether. On the East Coast of Mexico, again, the disease rises to a height of 800 to 900 metres (2500 to 3000 feet), as, for instance, at Cordova; while places above 1000 metres in height (3500 feet), such as Orizaba, Xalappa, and Puebla have hitherto escaped the sickness.[4] In Guiana and Venezuela the disease has always been confined to the coast belt; so that, as Zimpel (p. 78) remarks, one has only to take a short ride up country from Caracas to leave yellow-fever regions behind. Honda, near Bogota, at an elevation of some 200 metres (600 feet), as well as the whole of the plateau, has never been visited by the sickness, so far as we can tell from the data before us. In the interior of Brazil yellow fever must have been observed at least as far up as 700 metres (2300 feet), but there has never been an epidemic of it at Petropolis, which stands at a height of about 1000 metres (3500 feet), and at a distance from Rio of some five kilometres (three miles), notwithstanding the constant traffic with the capital

[1] Drake ii, 188, Dowler.

[2] Bellarin, p. 186, Carpentin, p. 47, Griffon du Bellay, p. 208.

[3] Report in the 'British Army Reports,' 1867, ix, p. 226.

[4] Bouffier, p. 526, Gouin, p. 404, Schmidtlein, p. 51, Heinemann (I), p. 161 (II), p. 156, Jourdanet, p. 208.

and the frequent importation of yellow-fever cases. Further, Constançia and San Paulo, situated at about the same height, have always escaped as yet.[1] In Spain the fever has attained an elevation of 300 metres (1000 feet), at the time when it was most widely prevalent in Andalusia; the occurrence of the disease in 1878 at Madrid (675 metres or 2000 feet in height) is a fact that stands by itself in the Spanish annals, and it was moreover an epidemic that was limited to fifty cases, of which thirty were fatal.

We cannot forget, at the outset, that the relative exemption of lofty regions from yellow fever, depends in part upon the kind of climate which they owe to their elevation, *i. e.* upon their relatively low temperature. But one or two considerations will convince us that this is not the only factor, nor even the principal one. In the first place the disease stops short at many points of the West Indies where the climate is still in the highest degree tropical; and that is true also of the town of Honda, whose temperature (28°52 C. or 84° Fahr.) is scarcely exceeded by any other place in Central America. On the other hand, there have been epidemics in cool weather at very considerable altitudes, as, for example, at Newcastle in Jamaica. I am rather inclined to think that the determining circumstances here are essentially the same as, or allied to, those that oppose the spread of the disease into the interior and to points remote from such water-ways as are large enough for ship traffic; and other obstacles would appear to be surmounted only exceptionally and under circumstances unusually favorable to the diffusion of the disease.

§ 88. An Urban Disease.

Another limitation to the area of yellow fever as an epidemic may be observed in the fact of its occurring almost solely in places with a crowded population; almost exclusively therefore in *towns*, and particularly in populous towns. On the other hand, country districts, even those in the immediate neighbourhood of towns, begin to be exempted so soon as they present the topographical and social conditions of the open country.

[1] Rey, p. 285, Lallemant, p. 12, McKinlay, p. 269.

"Yellow fever," says Drake (ii, 188), "is essentially a disease of towns large and small; the country people, even within a few miles of a town stricken with the disease, have nothing to fear from it so long as they keep outside of the sphere of the epidemic. The outbreak of a yellow-fever epidemic in a country district is an unheard of thing, although it has now and then happened that a few people living in the country near an infected town have fallen sick of the fever." La Roche (II, 335) speaks to the same effect: "In the country the disease never occurs, however constant and intimate the intercourse may be with the infected place. None are there affected but those who have taken the disease in the latter; and neither they nor such patients as are brought there from the city communicate the infection to anyone around them . . . On this subject the testimony of the profession is almost unanimous." The statements of observers from all parts of the yellow-fever zone bear out this last declaration most completely. The assertion may not hold good in its full extent; for example, yellow fever became prevalent in the epidemic of 1800 in a few villages and farms in the neighbourhood of Seville and Xeres,[1] in that of 1795 at two places in the neighbourhood of Newhaven (Conn.),[2] in 1839 in a circuit of some three miles around Mobile,[3] in 1853 at several villages and farms in Louisiana, Mississippi, and Alabama, as well as in Texas in 1859,[4] and in 1867 at two country estates in the neighbourhood of Pensacola.[5] But all these occurrences, as well as some of the same kind in Mexico,[6] took place at a time of very widely spread and severe epidemics, and the diffusion of the disease in the country remained for the most part strictly limited to the localities in question.

§ 89. Haunts the Low and Filthy Quarters of Seaports.

The more closely one inquires into the particulars of yellow fever, the smaller do the circles appear to be within which it has been prevalent at the various points of the yellow-fever zone; and these boundaries in space are drawn still closer, or the foci of the disease contract still farther, when we follow the malady into its several seats. In almost all the places where yellow fever has been prevalent hitherto—equally the indigenous fever and the imported—there are certain points from which the epidemic has always started; and those points are found to be, in seaports, the immediate

[1] Ferrari, Sonorampe.
[2] Monson, p. 177.
[3] Lewis (I), p. 289.
[4] Dowler.
[5] 'Philad. Med. and Surg. Reporter,' 1868, p. 228.
[6] Heinemann (II), p. 158.

neighbourhood of the harbour and the wharves, and, generally speaking, *the filthy quarters of the town*, the centres of poverty, misery and vice, with their narrow and foul-smelling streets, their tenements densely crowded from cellar to garret, their taverns, dancing saloons, and lodging-houses. It is after the epidemic has come to a head in those purlieus, that it begins to spread, always in the first instance into the immediate neighbourhood; but not unfrequently it remains confined to them, and the other parts of the town some distance off, and better situated hygienically, may be little troubled by the sickness or not at all.

"The places where the causes of the disease principally prevail," says Bone (p. 12), in speaking of his West Indian experiences, "are the vicinity of foul drains, the banks and channels of rivers which are dry at certain periods, the leeward openings of gullies, and crowded and ill-ventilated rooms and ships with foul holds;" and this statement is completely confirmed in the accounts of the local conditions under which yellow fever has been prevalent in Havana,[1] Bridgetown (Barbadoes),[2] St. John (Antigua),[3] Montserrat,[4] the ports of San Domingo[5] and Martinique,[6] in Castrie (Sta. Lucia),[7] Port Royal and Kingston (Jamaica),[8] Roseau (Dominica),[9] Spanish Town (Trinidad),[10] Nassau (Bahamas),[11] and in Bermuda.[12] In Georgetown (British Guiana) yellow fever has hitherto been limited as an epidemic to the filthy streets on the Demerara river, the scenes of poverty and misery.;[13] and the same conditions confront us not less strikingly in almost all the towns of North America lying within the zone of yellow fever. "We find," says Bancroft (p. 227), "that in New York, Philadelphia, Baltimore, Norfolk, and Charleston this fever always begins and often continues exclusively in the low streets immediately adjoining to the harbours and wharves of these towns, except in the case of some individuals, who, after having imbibed the noxious exhalations of the

[1] Barton, p. 369, Belot.

[2] Williamson, 'Med. Observ. relat. to the West India Islands,' Edin., 1867, i, 27. Ralph, pp. 55, 60.

[3] Musgrave, Furlong (I), p. 290.

[4] Dyatt, 'Med.-Chir. Rev. and Journ.,' iv, p. 1003.

[5] Desportes, I, p. 51, Gilbert, pp. 18, 19, Bally (I), p. 347.

[6] Savarésy, p. 174, Leblond, p 134, Chisholm (II), 78.

[7] Evans, p. 6, Levacher, p. 68.

[8] Hunter (I), p. 13, Jackson (II), Miller (II), Belcher, p. 247.

[9] Imray (I), p. 78, (II), p. 319.

[10] McCabe, p. 536.

[11] 'Brit. Army Reports,' 1864, vi, 255. [12] Smart.

[13] Chisholm (II), 200, Frost, p. 209, Blair (I), p. 33, account in the 'Lancet, 1867, ii, 200, 'Brit. Army Reports,' 1868, x, 69.

low streets in question, by residence or employment in or near them, happen to fall sick in other situations." For New York this statement is confirmed by Seaman (pp. 5, 34), Hardie (I, pp. 8, 28; II, pp. 2, 16), Miller (I, p. 99), Pascalis (III, p. 251), Watts (p. 302), Townsend (I, p. 111), Addoms (p. 7), and various others; the disease in that city has been limited mostly to the old and new slips, Pearl Street, Front Street, Water Street, and the surrounding quarter. For Philadelphia we have the evidence of La Roche (i, 46 ff; ii, 325, 362, 369) that the disease has always occurred in the first instance in the quarter of the city situated on the Delaware, and that it has not unfrequently been confined to that, or has appeared at the western and richer end of the city only in those streets which may be classed with the harbour quarter as regards overcrowding, dirt, and such like. In Baltimore the disease in every epidemic has first appeared on Fell's Point, a low-lying, damp, and filthy part of the harbour quarter, and it has never spread from thence except along the narrow dirty streets nearest to the shore, and has always stopped short of the high-lying west end of the city. We have this on the authority of Revere (pp. 220, 237), 'Letters and Documents,' &c. (pp. 15, 34), Potter (p. 21), Davidge (p. 66), Rush (p. 24), Moores, Valentin (II, p. 71), and others. In Norfolk (Va.) and Portsmouth (New Hamp.) it was likewise the densely tenanted filthy streets near the harbour that were always attacked, as we learn from Taylor (p. 150), Selden (I, p. 331), Archer (p. 61), Ramsay (III, p. 154), and from Bryant, who says (p. 295), after describing the localities: "The wonder is not that the pestilence prevailed, but that it has not annually swept the city, from the time these conditions began to exist." As regards Charleston, we have it from Sheeut (p. 100), Ramsay (I, p. 26), Dickson (II, p. 265), Lewis, Hume ('Charlest. Med. Journ.,' 1850, p. 29), Porter ('Amer. Journ. of Med. Sc.,' 1854, Oct., p. 342), and from the report on the epidemic of 1858, which all the physicians of the place agreed to, that the districts infested by yellow fever had increased just in proportion as the negligent public hygiene has permitted larger and larger quantities of refuse from the upper parts of the city to accumulate in the low-lying quarters. From New Orleans there come similar accounts by Chabert (p. 17, 23), Lemoine, Gerardin (p. 24), Thomas (I, p. 112; II, p. 59), Dowler (p. 42), and others; Barton (p. 348 ff.) calls attention to the fact that in the fourth district, whose sanitation was the most neglected, 7248 out of a population of 15,310, or nearly 50 per cent., sickened of yellow fever in the severe epidemic of 1858, or twice as many as in any other district of the city; and Fenner gives it as his conviction ('Southern Journ. of Med.,' 1866, May) that the exemption from yellow fever which New Orleans enjoyed at the time of the occupation by the Federal army (during the secession of the Southern States) is not to be ascribed to the blockade, which was often broken, but to the "despotically" conducted improvements in public sanitation. Events in Natchez have turned out in much the same way, according to Merrill (III, p. 217), Cartwright (I), and Monette (II, p. 75); in Memphis, according to

Drake (ii, p. 283) and Malloy (p. 343); in Vicksburg, according to Monette (III, p. 110) and Maegruder (p. 690); in Mobile, according to Lewis (II, p. 287), Drake (ii, p. 217, 219), and others; in St. Augustine, according to Strobel (p. 151) and Monette (II, p. 123); in Savannah, according to Fürth (p. 13), Waring (p. 11), Posey, and Woodhull (p. 23); Hill (p. 86), in Wilmington; Warren (II, p. 136), Wheaten (p. 333), Holt, Cain, Channing, and others, in Boston, Providence, and other towns of New England. Precisely the same circumstances meet us in the fever-stricken ports of Brazil,[1] Monte Video, and Buenos Ayres,[2] as well as in the epidemics that have occurred on Spanish,[3] Portuguese, or Italian soil. In Cadiz[4] the chief seat of the disease was always in the narrow, dark, filthy, and foul-smelling streets of the densely populated and poor Santa Maria quarter. It was the same in Gibraltar,[5] and in Seville, where, as we are told by Berthe,[6] the mortality in the epidemic of 1800 was about 5 per cent. in the clean and well-ventilated streets, but from 33 to 50 per cent. in the low-lying, damp, and filthy streets. For Malaga,[7] Xeres,[8] Barcelona,[9] and other towns we have similar accounts. Of the epidemic at Lisbon in 1723, we are told by Bancroft (I, p. 436), "The fever is very contagious in the lower parts of the city, going generally through a family, and very few families escaping it, especially in the close, narrow streets; the high parts are much freer than the low parts." And Lyons (p. 7) says of the epidemic there in 1875, "All the parts of the city largely attacked by the epidemic present in common certain conditions of insalubrity; defective water supply, total absence of, or more commonly extremely deficient sewerage, total absence of or incompleteness of house-drains and privies, and a consequently unclean state of the streets, badly constructed dwellings, &c." In Leghorn the epidemic broke out in certain narrow and filthy streets, and it did not spread beyond the surrounding quarter, inhabited by the proletariat and neglected in its hygiene.[11]

§ 90. Epidemics on Board Ship.

An interesting corollary to the circumstances here spoken of, and a fact of especial significance for the etiology, is furnished by the *epidemic prevalence of yellow fever on board*

[1] McKinlay, pp. 260, 345, Lallemant, p. 33, Dupont, p. 32, Rey, p. 281.
[2] Hiron, Scrivener (I and II), Morier, Lesson.
[3] O'Halloran, p. 179.
[4] Fellowes, p. 33, Berthe, p. 52, Arejula (I), p. 341, Doughty, p. 180.
[5] Bancroft, p. 473, Humphrey, p. 177, Amiel, p. 263.
[6] P. 162; see also Pariset et Mazet, p. 20.
[7] Fellowes, pp. 158, 166, O'Halloran, p. 179.
[8] O'Halloran, p. 141.
[9] Id., 6, Rochoux, p. 86.
[10] Palloni, p. 37, Lacoste, p. 43.

ship. These ship epidemics, of which a large number have been observed,[1] show the steps in the origin and epidemic continuance of the disease as if in a tableau—on a small scale, indeed, but just on that account the more easy to survey and to analyse in the details. The epidemics on board ship afford proof that the spread of the disease is absolutely independent of influences of the soil; they represent foci of disease within which the malady shows a preference, as on shore, for crowded, filthy, ill-ventilated holds, not unfrequently clinging to them exclusively, while its continuance on board ship is found to depend on the temperature of the air just as on land. Sometimes it has been only *one* cabin, *one* deck, or one *side* of the ship in which the disease chiefly raged, afterwards spreading to other quarters as well, and, under certain circumstances, even over the whole ship.[2] It is a noteworthy circumstance, and one often observed (as at Southampton in 1866 and 1867), that the officers and passengers of ships arriving with yellow fever at European ports had usually escaped altogether during the voyage, the cases of sickness having occurred among the sailors only.

"Nowhere more clearly than in the life of merchant seamen," says Lallemant (p. 29), "do we discover how essential in the last resort is filth as a cause of those attacks [of yellow fever];" and for proof he appeals to the spreading of the disease on board the filthy Finnish and Swedish vessels whenever these became infected.

It often happens that yellow fever shows itself first on board one or more ships lying in a port or roadstead of the yellow-fever zone, and does not become epidemic in the port itself until later; at other times it remains limited to the ships, without obtaining an epidemic footing on shore at all. In many cases it dies out—and this is especially apt to happen if the disease had been present on board in isolated

[1] See Rouppe, p. 68, Bryson (II), p. 181, Smith (VII). An excellent summary of the observations upon yellow fever made in the English navy during recent years is given by Friedel ('Die Krankheiten in der Marine, nach den "Reports of the Health of the Royal Navy,"' Berlin, 1866, pp. 102, 190, 208, 218, 246).

[2] See Fergusson (IV), p. 142, Wilson (I), p. 158, Pellarin, p. 188, Ricque, Forström.

cases only—as soon as the vessel leaves the port and gains the open sea;[1] while, in other cases, it continues as long as the ship's course is in low latitudes. Under these circumstances, it has often been observed that the disease has undergone a remission or even died out altogether when the ship has reached higher latitudes, and that it has been lighted up afresh as soon as she has come again into warmer regions. Under such conditions yellow fever has been enabled to continue on board ship for weeks and even months.[2] We shall afterwards see how prominent a part in the epidemic history of the disease such infected ships play.

§ 91. Geological Characters of the Soil of no Account.

The last important point in the inquiry before us is the question as to the influence that *conditions of soil* appear to have upon the production and diffusion of yellow fever. Whether the *kind of rock* has any significance in this respect cannot be settled offhand, in the absence of all exact inquiries directed to the point; this much, however, is certain, that the disease has become epidemic on the most various geological formations.

Wilson ('Memoirs,' pp. 89, 127) has pointed out that yellow fever, in its epidemic occurrence in the West Indies, appears to have been mostly associated with a chalk soil (belonging to the secondary formation), and he instances the prevalence of the disease in Jamaica, Trinidad, Martinique, Guadeloupe, San Domingo, and Barbadoes. But the disease has been observed not less frequently upon volcanic soil in St. Kitts, Guadeloupe, Dominica, Sta. Lucia, and Grenada. It prevails over a wide extent of the alluvial soil of Mexico, Brazil, Peru, and Guiana, and not less upon the diluvium and the tertiary formations[3] which extend from Texas along the southern and eastern coasts of

[1] Trotter (I), p. 358, Gillespie (I), p. 12, Morgan (I), p. 9.

[2] Gillespie (I), pp. 48, 53, Anderson (I), p. 21, Doughty, p. 16, Caillot, p. 199, Keraudren (I), p. 18, Moreau (I), p. 122, Lallemant, p. 115, Vanderpoel, 'New York Med. Record,' 1872, Dec., p. 82.

[3] Dowler points out that in the great epidemic of 1853 the disease was extensively prevalent on the high plateau with a diluvial soil which extends, under the name of the Bluffs, from Lake Pontchartrin, a long way up between the Mississippi and Pearl rivers, and which had been previously thought to be exempt from yellow fever. In the same epidemic the disease occurred also upon the

the United States as far as New Jersey. When the disease broke out in Spain, it was repeatedly epidemic upon chalk soil (in the hilly country of Cadiz), and upon Jurassic limestone (Valencia, Gibraltar, &c.), and even the oldest formations (granite and transition rocks) have not been exempt, in the outbreaks of yellow fever on the coast of Sierra Leone and of New England.

§ 92. Malarious Conditions of Soil are Irrelevant for Yellow Fever.

It has always been a subject of special interest to find out how far, if in any degree, yellow fever depends for its development and diffusion upon physical characters of the soil, upon the *porosity*, and *saturation*, and the *amount of organic matters contained therein;* how far the disease, in this respect, is allied to the malarial diseases. The inquiries directed to this point were for a long time vitiated by an error to which we have several times adverted in the course of these investigations; many observers have confounded yellow fever with malarial fever from the pathological point of view, and that is the chief reason why yellow fever has been pronounced to be *a disease of the soil* or *a swamp disease*, and has been ranged alongside the malarial fevers in its causation. It is only the experiences of the most recent times that have introduced a more rational conception of the problem and have effected a disentanglement that was much wanted; so that the number of those who at the present day adhere to the malarial theory of yellow fever—its adherents were chiefly found among American and English physicians—appears to be reduced to a very small figure, and the doctrine that yellow fever is dependent to some extent for its origin upon influences of the soil has been brought within proper limits.

Among the Lesser Antilles it is precisely those which, by reason of the conditions of their soil and especially of its swampy character, have been the principal seats of endemic malaria, that have been the least visited by yellow fever; whereas other islands, whose dry and rocky soil has prevented the endemic prevalence of malaria, have been

tertiary soils of Louisiana and Mississippi States at an elevation of 100 metres (330 feet) and upwards.

by far the most frequent scenes of yellow-fever epidemics. Fergusson, one of the best writers upon yellow fever, observes:[1]—"In the West Indies there are regions with as dry a soil and as free from swamp as are to be found anywhere on the globe; let us seek out the most favourable spots among them and send European troops there, and provided the place be on the coast, they will be decimated by yellow fever as surely as if we had sent them to Demerara or to any other of the most swampy places in the world." The same opinion has been expressed by Imray,[2] Anderson,[3] McLean (p. 25), Wilson (pp. 99, 129), and others with reference to Barbadoes (to which Fergusson had specially referred), St. Kitts, St. Vincent, Montserrat, and certain dry yellow-fever localities of San Domingo. From another quarter, Stewart (p. 186) in his report upon the yellow-fever epidemic of 1793-95 in Grenada, declares that the disease spared entirely just those places where malarial fevers prevail most. McCabe (p. 535) observes that yellow fever is comparatively rare on Trinidad,[4] one of the most marshy islands of the West Indies, and has been observed to be always confined to a few points, especially Port of Spain. Moreau[5] points out that the yellow-fever epidemics which were known to have occurred in Martinique up to 1820 had affected the two ports of St. Pierre and Fort Royal almost exclusively, while many places situated in the midst of swamps, some of them in the interior and some of them on the coast (for example, Port Marie in the south of the island), had escaped, the same being true of all subsequent epidemics. The disease has never occurred in the notorious swamp districts of the interior of Guiana;[6] and it would not be fair to attribute its endemic prevalence in Vera Cruz to marshy influences, for Jourdanet (p. 184), Crouillebois (p. 430), and others are unanimous in saying that the town suffers from malaria much less than the Téjeria, which is surrounded by marshes and is yet very rarely visited by yellow fever. "The Mexican coast," says Heinemann (II, p. 157), speaking generally, "affords numerous proofs that yellow fever is absolutely independent of so-called malarial fevers." The yellow fever of Charleston has never extended to the swampy districts of South Carolina, nor has it spread from Mobile over the damp flats of Alabama, or from Savannah over the rice-fields of Georgia; and, whereas malarial fevers have decreased remarkably in Charleston within recent years in consequence of improved drainage, yellow fever has continued up to 1871 as frequent and as malignant as before.[7] Bakel and Dabon, two notoriously malarious localities, the one in Senegambia and

[1] 'Edin. Med. and Surg. Journ.,' 1843, July, p. 186.

[2] Ib., 1845, Oct., p. 332.

[3] 'Facts,' &c., p. 16.

[4] Like British Guiana and Brazil, Trinidad has suffered much from yellow fever since that was written (1819).

[5] 'Memoire,' &c., p. 157.

[6] Bertulus (II), Blair.

[7] Gaillard, 'Transact. of the Amer. Med. Assoc.,' ii, 577.

the other on the Gold Coast, have never had an epidemic of yellow fever, although there have been often cases of the fever there, imported along the coast from Gorée and Grand Bassam respectively. In the epidemics of yellow fever which spread to a great extent over Spanish soil, it was precisely the elevated and dry-lying places that suffered from the sickness, while damp or marshy districts in their neighbourhood escaped; numerous examples of this might be adduced from Gibraltar, Medina Sidonia, Murcia, Xeres, and other places.[1]

A decisive argument in the question before us is afforded, on the one hand by the fact that yellow fever, in contrast to malaria, is met with properly in *towns* only, and quite exceptionally in country districts; and, on the other hand, there is the best argument of all in the *epidemic occurrence of yellow fever on board ship*. Chabert (p. 21) and Wilson (p. 89) are therefore right in stating that, if yellow fever occur at all in a marshy region, it is not the swampy soil but something else pertaining thereto that gives occasion to the development of the disease. At the same time, it should not be supposed that the soil, or the processes of decomposition occurring in it or upon it, are altogether without influence upon the production of yellow fever. Thus, it has been often observed that the breaking-up or turning over of the soil, the excavation of canals, the laying out of streets, embankments, and other such earthworks, appear to have had something to do not only with the outbreak of the disease, but also with its diffusion.

"For a century or more," says Jourdanet (p. 180), "the Spaniards who followed in the footsteps of the first adventurers to the New World do not appear to have died as the victims of the vomito negro. It was only after they had increased in well-being by clearing and cultivating the land, and had therein provided new guarantees for the maintenance of their health, that the yellow fever appeared among them, and spread widely over the newly drained localities." To the same effect is the opinion of Thomas ('Essai,' p. 72), on the outbreak and spread of the disease in Cuba and San Domingo. The first epidemic of yellow fever at New Orleans occurred, as Thomas (ib., p. 70) remarks, at the time when the channel of the Carondelet Canal was being excavated; Barton points out that many of the subsequent severe epidemics of yellow fever there (especially those of 1811-17-19-22-32-37-53) were coincident with canal undertakings, and that the same fact was observed at Francisville and other places in Louisiana in 1827-29-39.

[1] See Pym, pp. 31, 134, Bally (I), p. 355, Ferrari, p. 370, Pariset et Mazet, p. 75, Bally, François et Pariset, p. 460.

Accounts of the same kind come from Mobile, Natchez,[1] Charleston,[2] New York,[3] Trinidad,[4] Martinique,[5] Grand Bassam, and other places, several of the observers pointing out that the number of cases of sickness and of death from yellow fever reached its highest point in the neighbourhood of the places where the earthworks were being made, and among the labourers who were employed upon them.

§ 93. Nature and Origin of the Morbid Poison.

For yellow fever, as for all infective diseases, a specific and material morbid poison must be assumed. We must abandon, as misdirected, all attempts to discover the genesis of the disease in the action of atmospheric forces—in high temperature, either by itself or in association with extreme degrees of atmospheric moisture, great electrical tension of the atmosphere, and the like. But while there can be little doubt as to the material and specific character of the morbid poison, the precision hitherto reached in the views about the nature of the poison has been just as slight in this, as in nearly all the other infective diseases.

It is highly probable, for several reasons mentioned above, that the development or multiplication of the yellow-fever poison stands in a certain causal connexion with processes of decomposition in organic matters; and thus we are confronted here with the same question as in malaria and all other acute infective diseases: whether namely, the disease-producing factor is to be sought for among those decomposition-products themselves, or in whatever sets the decomposition agoing, or in other organic (or organised) forms standing in a certain relation to the processes of putrefaction.

Hypotheses as to the *nature of the yellow-fever poison*, leaning sometimes to one side, sometimes to the other, have exhausted the ingenuity of the profession without advancing our knowledge by a single step.

[1] Merrill, 'Philad. Journ. of Med. and Phys. Sc.,' 1824, ix, p. 240, and 'Memphis Med. Recorder,' i, 87; Barton, p. 317.

[2] Simons, 'Charlest. Med. Journ.,' 1853, viii, p. 363; Wragg, in La Roche, ii, p. 405.

[3] Bayley, 'Account,' &c., p. 59.

[4] McCabe, p. 535.

[5] Ballot, 'Arch. de méd. nav.,' 1870, Jan., p. 61.

One of the boldest hypotheses as to the septic nature of the poison is that which has been elaborated by Audouard.[1] In his opinion the poison of yellow fever has been engendered in the crowded, filthy, unventilated holds of slave-ships. They were the first means by which the sickness was ever brought to the West Indies and the mainland of America. Although the poison had not proved dangerous to the negroes themselves, it clung to the ships, and had been repeatedly taken to Europe on the voyage back with a cargo of merchandise. This view of the importation of the disease by ships conveying negroes is also applied by him to explain the more recent general outbreak of yellow fever in Brazil.[2] Some observers have hazarded the conjecture that the yellow-fever poison is generated out of the putrefaction of certain marine animals, especially the madrepores, which accumulate in large quantities on the shores of the West Indies and of the Gulf of Mexico.[3] The view that the origin and reproduction of the poison is specially related to timber has been particularly well received. Fergusson[4] was the first to apply it to explain the frequent occurrence of the disease on board ships, and their tenacity of the poison. After him Rochoux, and more especially Wilson,[5] pointed out that it was just the ships with cargoes of timber that chiefly became foci of yellow fever; and Wilson at the same time directed attention to the fact that, on shore also, the disease occurred with especial frequency at places where there was much decaying wood, as on wharves, docks caissoned with wood, and ships' moorings. This view has found supporters in Bryson,[6] Bush, and others. In other quarters special weight has been laid on certain cargoes, particularly sugar and coal;[7] while others again have attributed the direct cause of epidemics on board ship to the decomposition of the organic matters contained in the bilge-water.[8]

In the most recent times, investigators have inclined more to the theory of a parasitic origin of the disease,[9] although no one has yet succeeded in adducing a single positive fact upon which to base this theory. It is clear that the assumption of a parasitic origin for yellow

[1] 'Revue méd.,' 1824, Nov., iv, 227.

[2] Ib., 1850, July, ii, 67.

[3] Maher, 'Relat. méd. de deux épidémies de fièvre jaune à bord de la frégate d'Herminie, 1817–38,' &c., Paris, 1839, p. 131; Bertulus (II); Dupont, p. 57, and others.

[4] 'Med.-Chir. Transact.,' l. c.

[5] 'Memoirs,' &c., p. 139 ff.

[6] 'Report,' &c., p. 229.

[7] The disease has, in fact, occurred remarkably often in sugar ships and coal ships, which are distinguished in general from other merchant ships by want of cleanliness. See Townsend, in the 'New England Journ. of Med.,' xii, p. 381; Buckley, 'New York Journ. of Med.,' 1856, Sept.; Mélier, pp. 256, 301.

[8] Archer, p. 61, Barrington, Maccoun, p. 325, Gibbs, p. 344, Rieque, Jewell (I), Hommel, p. 7, Schmidtlein, p. 51.

[9] Pellarin, Mélier, Schmidt, Gaillard, Gibbs, Macdonald in the 'Statist Rep. on the Health of the Brit. Roy. Navy, 1860,' p. 77, Nott, Sternberg, and others.

fever is incompatible with the *spontaneous origin of the disease*; and it is in this sense that Macdonald, one of the most decided partisans of the parasitic theory, expresses himself when he says: "There is no more proof of the spontaneous development of a monad than of an elephant; the doctrine of the spontaneous origin of the yellow-fever organism or cause, if this premise be sound, can have no foundation to satisfy the rational mind."

§ 94. The Question of Communicability.

But this want of acquaintance with the nature of the yellow-fever poison does not prevent us from obtaining a tolerably clear insight into the *way in which the disease spreads;* including the question whether the native habitat of yellow fever extends as far as the area of its distribution, or whether it is endemic at only a few points on the globe, occurring elsewhere merely in consequence of importation of the morbid poison; and, in the latter case, we have to inquire by what media and in what ways that importation takes place.

The question so keenly discussed a good many years ago as to the *communicability of yellow fever* has been definitely answered in the affirmative by the most recent events in Brazil and Peru, at Lisbon, Barcelona, and elsewhere; and if there are still some differences of opinion among the observers, it is not now a question whether communication actually takes only, but only of how it takes place. And those differences depend not so much upon conflicting facts, but rather upon the significance ascribed to them; and they arise above all from the futile endeavours to interpret the facts according to our conventional and obscure notions of "contagium" and "miasma." If we are to understand by *contagion* that kind of spreading of a disease in which we are concerned with infection of an individual by direct conveyance of a morbid poison reproduced in a specifically diseased organism and eliminated therefrom, then, for yellow fever, that mode of disease-conveyance has to be at once dismissed. It is at variance with the character of a "contagious" disease that it should prevail, as in the case of yellow fever, almost exclusively among the population of the coast or river shore

and indeed among urban populations, that it should occur only exceptionally under other circumstances of locality, and that it should almost entirely spare not only the country population who are engaged in open traffic with the infected place, but the interior in general, and that too in spite of the introduction of numerous cases of the disease, especially among the large numbers of fugitives from the infected towns who resort to the country.[1] In very many cases, yellow-fever patients have been landed from ships without the disease being communicated round about, whether in the hospitals or in the private dwellings into which they have been admitted. Even the most intimate kinds of contact, such as the healthy and the sick sleeping in one bed, the attendance of physicians and nurses upon the sick,[2] the use of the uncleansed linen, clothes, or beds of yellow fever patients,[3] post-mortem examinations of their bodies,[4] and the like, have in no wise contributed to the spread of the disease. Particular emphasis has been laid in some quarters upon the fact that specially designed experiments to induce infection by the inunction or inoculation into the skin of the vomit of yellow-fever patients, and by the wearing of the linen and clothes used by the sick and saturated with their perspiration, have always yielded a negative result.[5]

The experiences of the frightful yellow-fever epidemic of 1878 in North America have proved highly instructive in this respect. There is but one opinion as to the non-contagiousness (in the strict sense) of yellow fever, held by those

[1] See the accounts of Lining and Ramsay (VI) from Charleston; of Smith (IV), p. 109, Bayley (II), p. 126, and Miller from New York; of Rush (I), p. 132, and Devèze, p. 220, from Philadelphia; of Bowen (II), p. 338, from New Providence; of Chabert, pp. 21, 97, Gros et Girardin, p. 7, and Thomas (I), p. 144, from New Orleans; of Monette (II), p. 75, from Natchez; of Goupilleau from Tampico; of Baker, Lallemant, and others from Brazil; of Mantegazza from Buenos Ayres; of Boyle, p. 291, from Sierre Leone; and of Guyon (II), p. 441, from Lisbon.

[2] See Clark (I), p. 22, Leblond, p. 226, Potter, p. 21, Vaughan (I), p. 371, Dunlop, Porter (III), O'Halloran, p. 102, Lacoste, Fürth, p. 17, Scrivener (I), Sternberg, and others.

[3] Valentin, p. 122, Devèze, p. 221, Birnie, p. 336, Barrington, p. 310.

[4] Devèze, p. 239, Gillespie, 'Observ.,' &c., p. 69, Rochoux, 'Recherch.,' &c., p. 315, Doughty, p. 49.

[5] Rochoux, l. c., Devèze, p. 13, Dupuy, Guyon after a statement by Donzelot.

who have published accounts of that epidemic (Stillé, Lynch, Thomas (VI), Whittaker, Woodworth, Bayley (IV), and others); the scientific commission appointed to inquire into it have reported to the same effect;[1] and that may be said also to be the conclusion recently arrived at by Lawson after a very thorough inquiry into the spread of the disease on the Sierra Leone Coast and in the West Indies, both on land and on board ship. Lawson's opinion is summed up in these words:—"Healthy persons going into a locality where the cause [of the disease] was known to be in a state of activity were very liable to be attacked, but if they or others infected in such a locality returned to a healthy one, they went through the fever there without communicating it; there was no evidence to show that persons labouring under the yellow fever, or the bodies of those who had died of it, gave off a poison capable of exciting the disease." This passage may be taken to express the most recent conviction arrived at by the whole of the medical world acquainted with the circumstances in question.

But if the question concerning the contagious character of yellow fever is decisively answered in the negative, just as certainly do all the facts point conclusively to *communicability of some kind*. Setting aside certain of the West India Islands, a part of the Mexican Gulf Coast, and the coast of Sierra Leone, we find that at all other places where yellow fever has ever been prevalent, the outbreak of the epidemic, or even the occurrence of isolated cases of the disease, has *always* followed shortly after the arrival of ships from ports where the disease was known to be epidemically prevalent at the time of their sailing,[2] or which were notorious as endemic foci of yellow fever.[3] In most cases, the ships have had yellow

[1] 'New York Med. Record,' 1878, Nov., p. 434.

[2] It may be mentioned here that the carelessness of sanitary authorities at ports of arrival has played a very disastrous part in the history of yellow fever, especially in the last century. The mischief being done, those officials often attempted to envelop the facts in obscurity, and thereby to cover themselves. They would bring forward evidence to show that cases of yellow fever had occurred before the arrival of the infected ships, and so forth.

[3] "In the West Indies one should never trust," says Friedel (p. 224), "the rumour that there is no yellow fever. A ship of war is always a mine of wealth to the inhabitants of these colonial ports, and they are careful to keep silent about the sporadic cases of yellow fever that occur from time to time, so as not

fever on board on their arrival, or have had it on board during the passage; but there are also well authenticated cases in which such an importation of the disease has taken place from ships which had sailed from infected ports, but on board of which no yellow-fever sickness had occurred, either among the crew or among the passengers.[1]

The causal connexion between infected ships and the outbreak of the disease at the ports where they have arrived comes out still more decidedly in those frequently observed instances where the first case of illness, sometimes even a whole series of cases, has originated on board the ship itself among custom-house and quarantine officers, pilots, ship labourers, and others, or where the starting-point of the infection has arisen through indirect intercourse with the infected ship, by means of goods, ballast, stores, clothing of the crew, and the like unladed from it.[2]

At the present day, we cannot longer doubt that the instances of yellow fever in European ports, the outbreaks of it on the coast of Senegambia,[3] and in the islands off the West African coast, the epidemics of it on the continent of

to interrupt the shipping traffic and their own business." What is here said of ships of war applies equally to merchant ships, as we may learn from numerous accounts; in fact, cases of the latter kind are met with more frequently, inasmuch as there is often a want of a due degree of caution on the part of the captain.

[1] Cases of that sort have been published by Hohenberg from New York, and by Jewell (I) from Philadelphia. One of the most interesting cases occurred in the recent outbreak of the disease at Madrid in 1878. Among a force of 17,000 to 18,000 troops returning from Cuba, not a single case of yellow-fever sickness occurred either on the voyage or subsequently. The troops disembarked at Santander, and about one half of them were sent by rail to Madrid along with their unopened baggage. The baggage was not unpacked and cleansed until it reached the capital, whereupon it gave rise to an outbreak of the disease. The soldiers themselves, who were, to be sure, acclimatised, remained exempt from it. "The yellow fever at Madrid," says Guichet, "was due to the importation of morbid germs among the clothes and baggage of men in good health returning from Cuba;" and the writer in the 'Lancet' (1878, Nov., p. 641) expresses the same opinion.

[2] The first cases in the New Haven epidemic of 1794 occurred in individuals who had been employed on board a vessel from Martinique in removing the clothes of several persons who had died of yellow fever on the voyage (Monson, p. 174; 'Additional Facts,' &c., p. 50).

[3] Fergusson had stated long ago ('Lond. Gaz.,' 1839, p. 839) that yellow fever occurred in Senegambia only after importation from Sierra Leone; and that opinion has been subsequently confirmed by Thevenot (p. 254), Cedont, and all later observers.

South America, as well as the cases that occurred previous to 1820 in some New England ports, have always been associated with the arrival of infected ships; and it is now equally beyond doubt that in New York, Philadelphia,[1] Baltimore, Norfolk, and other seaports of the Central States of the Union, yellow fever has never occurred without an infected ship having arrived just before. This fact has been longest in getting accepted for the United States ports on the southern part of the Atlantic coast and on the Gulf coast; but for these also, particularly Charleston, Savannah, the Florida ports, and Mobile, the conviction has almost everywhere gained ground that every outbreak of yellow fever in them (apart from exacerbations of slumbering epidemics such as have occurred also in Spain and South America) may be brought into connexion with the arrival of vessels from suspected ports.

"It is difficult," says Dickson ('Charlest. Journ.,' 1856, p, 749), "to argue with a man who can now doubt the communicability of this pestilence, its transportation, or the transportation of its cause, from place to place; . . . . Whether it occurs in New York or Boston, Philadelphia or Baltimore, Norfolk or Charleston, Savannah or Mobile, it is always coincident with or subsequent to some foul arrival from the West Indies or New Orleans, perpetual sources, as I suppose will be admitted, of the pestilence."

In the most recent times, and especially since the experiences of 1867-78, scarcely a doubt exists that even in New Orleans the disease is not endemic but always imported, and that yellow fever, therefore, must be regarded as a disease everywhere exotic to United States soil.[2]

The history of *yellow-fever epidemics on board ships* themselves furnishes an additional and important contribution to the doctrine that the disease spreads by way of communication. Ships have got yellow fever only when they have been in direct or indirect intercourse with other

[1] The attempt made by La Roche to show that the appearance of yellow fever in Philadelphia had not depended upon the importation of the disease by ships has quite miscarried, inasmuch as the author has confounded yellow fever with bilious remittent malarial fever.

[2] Woodward, 'Report on Epidemic Cholera and Yellow Fever in the Army of the United States during the year 1867,' Washington, 1868; Stillé, Woodworth, and various others.

ships already infected, or with an infected shore; and there is not in the whole literature of yellow fever a single accredited instance of the occurrence of the disease on board a ship voyaging to, or moored within the yellow-fever zone, in which that condition has not been fulfilled.

So long, says Fraser ('Lond. Med. Gaz.,' 1838, xxi, p. 642), as a ship lying in a yellow-fever port keeps clear of all communication with the shore, no case of yellow fever will show itself on board; but the strict rules adopted by captains in these cases seldom continue to be observed longer than the first week or two, after which time some one or other of the crew steals ashore to visit a friend or to make a purchase, and, with that, the ship's protection from the sickness is gone. Friedel also points out how often, in ships of war, the disease is brought on board by those sailors who row the officers ashore, and have often to wait for hours to row them back. A most complicated case of the transmission of yellow fever from one ship to three others, and to the shore, is related by Bryson ('Epidemiol. Trans.,' i, 187). A vessel having been infected on the coast of Mexico or of San Domingo, brought the disease first to Port Royal, and came into communication there with the crew of a second ship, on board which cases of sickness occurred shortly after; from the latter a third was infected through surplus hands being transferred, while finally the disease was conveyed from the first ship to another (the fourth), and at length to the hospital of Port Royal.

We find a complete analogy for these ship epidemics in the behaviour of the disease on land within the infected towns. An analysis of any yellow-fever epidemic shows certain groups of cases so arranged as to constitute separate foci of disease, sometimes in single houses, sometimes in blocks of houses, or, again, in streets or groups of streets; so that each new case of disease, as it occurs, may be traced to infection of the individual within any one such focus. That case may in its turn become the centre of a new focus; but outside those centres of disease there is complete immunity.

The importance of these foci of infection for the diffusion of the disease had been already rightly appreciated by many observers in the latter half of the last century—by Monson (p. 177), McKnight (p. 293), Addoms, Steward (p. 123), Chisholm (II, pp. 96, 107), Warren (p. 136), and others. Observations to the same effect were published for the Spanish epidemics by Fellowes (p. 287), Bally (p. 82), Pym (pp. 20, 47), and almost all the other chroniclers. Guyon (II, p. 441) remarks concerning the Lisbon epidemic of 1857: "Once in a house, the scourge will always carry off a larger or smaller number of victims from it; further, *whether there are still sick persons in it, or whether there are no*

longer any, that house will become a centre to reproduce the disease in the strangers who enter it; and what is true of a whole house applies also to a part of it." Many observers have reported to the same effect from North and South America, the West Indies, and Mexico; and even Blair, one of the keenest opponents of the doctrine of the communicability of yellow fever, cannot but admit that the disease is always confined at first to single foci, from which it spreads and forms new foci of infection, and he adds: "*Outside these boundaries of epidemic influence there was safety.*"

§ 95. Goes no farther than Maritime Commerce goes.

The reason for the limitation in space of a disease intrinsically communicable—for that is very markedly the character of yellow fever—must be sought for, assuming that there is free traffic between infected and healthy places, either in certain local conditions, whether of the attacked or the exempted locality, or in the media by which the communication of the disease is effected, or finally in a peculiarity of the morbid poison itself. As regards the last point we are not able to say anything at all; and even with reference to the first, an impartial and thoroughly objective review of the facts affords nothing on which an explanation of the circumstances in question might be based. Neither in conditions of temperature, of soil, of social life, of hygiene, or of population are the differences so considerable, on the one hand, between the interior plains of Louisiana, Mississippi, Alabama, and other Gulf Coast States, and their shores on the other, that we should explain thereby the almost uniform exemption of the former from infection; although it should not be forgotten that differences may exist of a kind that have hitherto entirely escaped our attention. There is but *one* fact always presenting itself wherever we turn our eyes, both within the yellow-fever zone and outside of it, and that is the close connexion of the disease with sea coasts and the shores of navigable rivers. It is here that we meet with the factor which appears to have direct control over the spread of the disease, viz. *sea voyages,* or maritime commerce. *Yellow fever goes practically no farther than the traffic by sea; the disease for the most part finds its limit*

where that medium of communication ends. We are thus led to assume that the morbid poison clings to the ships themselves, or to the goods, chattels, or persons which the ship carries, that it is taken up by ships from its indigenous or its epidemic foci, and transported to other places, where it gets the opportunity, under the conditions above mentioned, of putting forth its power. There are numerous well-authenticated observations proving beyond doubt that ships under those circumstances may become foci of infection, within which a crew made up of acclimatised persons may live without taking harm; they do not make their injurious influence felt until susceptible individuals come on board in a distant port and enter the infected holds. In the same way a complete explanation appears to be furnished of the often observed fact that, where an infected ship is placed under strict quarantine, only those inhabitants of the port take the disease who have for one reason or another gone on board. If the rest of the population sickens, if new foci of infection are formed in the port, and the disease thereby becomes an epidemic on shore, then we must further assume that the morbid poison has been conveyed to land; and this can only have taken place either by the wind blowing from the vessel shorewards, or by personal intercourse, or by traffic in goods.

§ 96. Alleged Diffusion by the Wind.

That *the wind may be a carrier of the yellow-fever poison* cannot *à priori* be denied; and it may indeed be taken for granted that the diffusion of the disease to short distances does take place in that way. On the other hand, none of the observations claiming to prove a diffusion of the disease over wide tracts by currents of air are to be regarded as conclusive, inasmuch as, in all those cases, a conveyance of the morbific cause from place to place in other ways is not only not excluded, but is much the more likely thing to have occurred.

Chisholm (I, p. 311) estimates the distance to which yellow-fever poison may be borne by the wind at about six to ten feet; others put it at a quarter of a mile and even more. In Strobel's account of the

epidemic of 1839 at Charleston,[1] there is a statement according to which the extension of the disease from one vessel to three others lying at a distance of a quarter to half a mile took place by the wind. Milier reports a similar fact in the well-known incident of 1861, in the harbour of St. Nazaire: of the ships in the neighbourhood of the infected craft, those only were attacked by the sickness which lay to the leeward, while those that anchored to windward escaped. It has been observed on various occasions, as for example in 1848 at New York, according to Bodinier, and at Philadelphia in 1858, according to Jewell, that only those of the population were affected with the disease who lived close to where the infected ships were berthed, and these only in so far as they were exposed to winds blowing across the ships. According to the official account of the epidemic of 1855 at Norfolk, the town was infected from the suburb of Gosport by the wind; Jaspard and Potter (p. 20) assure us that in Baltimore one could always predict the march of the disease from the prevalent direction of the wind. All these data, and others like them, deserve little confidence, inasmuch as the observations are not free from error. In the instances cited from New York and Philadelphia, the persons living upon the quarantine ground, where the later cases of sickness occurred exclusively, had communicated with the infected ships directly or indirectly. Whether any communication had taken place among the ships in the anchorage at Charleston in 1839 is not mentioned; but Milier expressly denies it, at all events, for the ships in the harbour of St. Nazaire. The extension of the disease from Gosport to Norfolk is much more easily explained, according to Williman, by the free traffic between these places by means of a steam ferry. The account by Cummins[2] of the disease being carried by the wind from the shore to a ship of war lying in the port is an instance of the scanty criticism that has been brought to bear on the discussion of this question; the crew were strictly forbidden to land at the infected port, only the officers being allowed to go on shore at pleasure; and yet the disease broke out on board the ship, not among the officers but among the crew. But nothing is said of the sailors who rowed the officers ashore and waited to row them back.

In deciding on this question, we have to remember, firstly, that any such conveyance of the yellow-fever poison by the wind from ships to the shore or to other ships, or conversely from the shore to ships, is, at all events extremely rare, if it occur at all; secondly, that the very narrow limits of the foci of infection[3] indicate a high degree of tenacity or a considerable specific gravity of the poison; and thirdly, that

[1] 'Essay,' &c., p. 23.

[2] 'Lancet,' 1853, July.

[3] Heinemann has called attention to this fact in his experiences at Vera Cruz and neighbourhood.

many observers have known the disease to spread regardless of the direction of the wind and even against the wind. No real importance, therefore, can be attached to the wind as a carrier of the disease to any considerable distance.

§ 97. Diffused by Personal Intercourse or Goods Traffic.

There remain, then, only *the intercourse of individuals, or the traffic in goods, as efficient means for the diffusion of the disease;* and, as a matter of fact, the history of yellow fever has at all times afforded, as we have seen, the most incontestable proofs that persons or effects, coming from a focus of infection, not unfrequently bring with them that which gives rise, under certain circumstances, to the formation of a new centre of sickness; while the multiplication of such centres of infection amounts to an epidemic. At the outset of this inquiry into the ways in which the disease spreads, I touched on the facts that tell against a diffusion by contagion proper, or against a reproduction of the morbid poison in the bodies of the sick. I hold it to be beyond question, accordingly, that the yellow-fever patient is a medium of spreading the disease only in so far as the morbid poisons clings to him as it does to other objects; that, in this respect, he plays no other part than his effects, or the coffers in which he keeps them, or than ships utensils, bales of goods, ballast, and in short, all that a ship carries, including perhaps the ship's hull itself. I hold also that the same applies to the formation of disease-foci on shore;[1]

[1] There are numerous accounts of the disease having been spread by infected linen, luggage, and other personal belongings: as, for example, those of Pariset et Mazet (pp. 89, 91) for the Cadiz epidemic of 1819; of Bally, and of François et Pariset (p. 82), for the 1821 epidemic in Catalonia; the official report of the epidemic of 1819 at New York ('Amer. Med. Recorder,' iii, p. 203); that of Palloni (p. 36) for Leghorn; of Harris ('Report of the Physician-in-chief of the Marine Hospital at Quarantine,' Albany, 1857) for New York in 1856; of the Portuguese physicians ('Relatorio,' &c., Lisboa, 1859) for the Lisbon epidemic of 1857; of Rochester, Thomas, Vance, and others, for the epidemic of 1878 in the United States. As to the morbid poison clinging to rooms, the following interesting fact comes from Port's Island, Bermuda, under date 1869 ('Lancet,' 1869, Oct., p. 583):—The naval authorities had told off a number of sailors to clean out the quarantine hospital, and the work had just been begun, when a

that the potency and renewal of the morbid poison is dependent on certain influences without (the temperature and the amount of moisture), and that it would seem to be essentially connected also with the processes of decomposition in organic matters. At the same time, *the limit to the diffusion of the disease by way of the land traffic is soon reached.* Notwithstanding the most careful scrutiny of all the factors herein concerned, I have been unable to discover any reason for this peculiar limitation, which is without analogy in the history of other acute infective diseases. We are confronted here with one of those riddles, of which the history of the acute infective disorders furnishes so many, and of which there will probably be no explanation until we succeed in becoming better acquainted with the nature of the morbid poisons themselves.

§ 98. Endemic at Three Points Only.

It has been abundantly shown in the course of these inquiries that *the native habitat of yellow fever* is not so extensive as the area of its diffusion. These inquiries should have enabled us to define the endemic foci of yellow fever somewhat precisely; we may take them to be those places where the disease occurs habitually in sporadic cases, and where an epidemic may spring up from such sporadic cases and independently of any imported sickness.

From that critical standpoint, then, we find, throughout the whole of the yellow-fever region, only *three* situations at which the disease bears an undoubtedly endemic character —the West Indies, the Mexican Gulf Coast, and part of the Guinea Coast. The headquarters of the disease are unquestionably the *West Indies,* more particularly the Greater

ship arrived from the West Indies with yellow fever on board. As the sick from this vessel had to be taken into the hospital, the cleaning was put off for a time, and the sailors ordered back. Twenty-seven days after the last convalescent had left the hospital to go on board, and the vessel had sailed again for Halifax, the sailors, eight in number, resumed the work of cleaning, and within the next eight days two of them sickened and died; a third fell ill six days later; and the remaining five men were thereupon sent to Halifax on board a sloop-of-war, under the charge of a surgeon, who himself took the fever and died.

Antilles,[1] from which came the first reliable accounts of yellow-fever sickness among the European emigrants to the Western Hemisphere. Heinemann thinks himself justified in concluding from his inquiries [2] that the disease is endemic at only five places on the Mexican part of the Gulf Coast, viz. at Vera Cruz, Alvarádo, Tlacotálpam, Lagúna, and Campéche; but even in these, he adds, it is not endemic as an original malady, but as one naturalised after repeated importations from Cuba. Finally, on the *Guinea Coast*, the endemic focus of the disease appears to be exclusively Sierra Leone. Against the notion started, with no reason at all, by Pym and others, that this was the proper home of yellow fever,[3] and that the disease had been imported thence to the West Indies, it may be urged that the first accounts of yellow fever on the West Coast of Africa do not date back beyond the eighteenth century, and that it is much more probable, therefore, that this region was infected from the Antilles, afterwards becoming an endemic focus when the disease had got naturalised. Whether that endemic focus of disease includes other regions of the Guinea Coast as well, is at least very questionable; the disease has always been an importation on the Congo Coast, the coast of Senegambia, the West African Islands, and, in fact, at all other places in that part of the world which have been visited by yellow fever at all. The same is true of the few regions of Europe that have been attacked by it; and it is further true, as we have already seen, for the continent of North America, and most probably also for that of South America, particularly for Guiana, Venezuela and Peru. Finally, as to the position of the

[1] It is difficult to decide whether endemic foci of yellow fever occur in certain of the Lesser Antilles, owing to the close intercourse that they all keep up with one another. The endemicity of the fever is denied for Martinique and Guadeloupe by Leblond (p. 133), Rufz (II), Dutroulau ('Traité,' &c., pp. 34, 362), and St. Vel; for Barbadoes by Jackson ('Boston Med. and Surg. Journ.,' 1867, July, p. 447); for Antigua by Furlong and Nicholson; for Grenada by Chisholm (I, p. 96); for Trinidad by Leblond (p. 136); and for Nassau (Bahamas) by the military reports ('Brit. Army Report' for 1864, vi, p. 255). On the other hand, we are entitled to consider St. Thomas, Santa Cruz, Santa Lucia, and St. Vincent as endemic seats of yellow fever, on the authority of Leblond, Gartner, Miller (III), and others.

[2] (II), p. 140.

[3] Hence the equivalent "Bulam fever" for yellow fever.

disease in Brazil, a decided opinion cannot be given off hand. It appears that yellow fever, since it first appeared in that country in 1849 and 1850, has never died out; but whether that persistence for thirty years depends upon repeated importations of the morbid poison or upon the formation of endemic foci, cannot be decided at present, as there has been no thorough-going inquiry into the subject.

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CHAPTER IX.

ASIATIC CHOLERA.

§. 99. First Pandemic, 1817-23.

In the nineteenth-century annals of pestilence, the year 1817 stands as one charged with fatality to the human race. It was in that year there began the epidemic extension over India of a disease which had previously been known only as an endemic in a few districts of the country; in that and the following year it overran the whole peninsula, in a short time it crossed the borders of its native territory in all directions, penetrated in its farther progress to almost every part of the habitable globe, and thus acquired the character of a world-wide pestilence, which has repeatedly since then entered on its devastating campaigns and has claimed its many millions of victims. This sickness, known in its native seats under the names[1] of *Morshi*, *Mordeshin*, and *Visuchika*, having had afterwards the name of *Cholera* given to it, from a form of disease nearly related to it in symptoms, and the name of *Asiatic cholera* (or preferably *Indian cholera*), to distinguish it from the allied forms, has, up to the present, broken out and spread to the extent of a pandemic, on Indian and extra-Indian soil, four successive times. The first pandemic comprehends the period from 1817 to 1823, the second from 1826 to 1837, the third extends from 1846 to 1863, while the fourth began in 1865 and ended in 1875.

The point of departure of the first pandemic in 1817 is enveloped in impenetrable obscurity. It appears that the disease had already begun be epidemic in 1816 in a district of Behar, in the neighbourhood of Purneah.[2] The first reliable information as to its diffusion dates, however, from the spring of 1817.[3] In the month of May it was at

[1] See Macpherson ('Annals of Cholera,' Lond., 1872, pp. 6—14) for the numerous synonyms of Asiatic cholera in its native and foreign seats.

[2] Conf. Macpherson, ib., p. 151.

[3] For the spread of cholera in the years 1817-19, see especially the 'Report on

Kishnaghur, on the Hooghly, in June at Maimansing, on the Brahmaputra, in July at Patna and Dacca (in the lower basin of the Ganges), in the beginning of August at Calcutta, and in the middle of that month at Jessore. Almost simultaneously with its appearance in the last-mentioned locality, where the attention of the authorities was first drawn to its epidemic character, it showed itself in the districts of Chittagong (eastern shore of the Bay of Bengal) and Rajashahja (valley of the Ganges); in the beginning of September it came to Bhagulpoor and Mongheer, in the middle of that month to Purneah, Dinajepore, Buxar, and Ghazipore, and in the beginning of October it was in the districts of Berhampore and Rungpore. So that, in the space of four months, the pestilence had already overrun a great part of Lower Bengal. Early in November the epidemic broke out in Mirzapore and in the Bundelkhund State, where it was prevalent especially among the English troops under Hastings, and penetrated as far as Riva. During the months from December to February, the sickness appears to have been everywhere extinguished, or at least to have remitted materially; but in March of 1818 it reappeared at most of the places where it had already been, and now it spread over the whole of Hindostan, a great part of the Deccan, and along the eastern and western seaboards of India; so that, in the course of that year, only a few of the larger tracts of the country, more especially the hill districts, escaped it. The epidemic broke out in March of that year at Allahabad (at the confluence of the Ganges and Jumna) and in Bundelkhund, whence it spread in a north-westerly direction along the plain of the Ganges, by Agra, Mattra, Delhi, Oudh, Lucknow, and Bareilly, travelling through the North-West Provinces to the Punjaub; and in a westerly and south-westerly direction by Malwa to Khandeish, reaching Goojerat and the Raja states, and thereafter Aurangabad and Poona, until it came in August to Bombay, whence it overran the Konkan and extended to the Malabar Coast. From Lower Bengal the pestilence had in the meantime penetrated into the hill districs of Tirhoot (where it attained to an elevation of 2000 feet and upwards), and it had at the same time taken a southerly direction towards the Presidency of Madras, had reached Ganjam by the month of March, was at Vizagapatam in May, at Masulipatam in July, at Nellore in August, at Madras in October, and at Pondicherry a little later; while in the meantime it had spread from the coast into the interior, visiting Hyderabad, Bellary, Arcot, Bangalore, Seringapatam, Mysore, and the Salem districts, whence it extended in the beginning of 1819 to Palamacotta, one of the most southern points of the Carnatic. For Sind and the Punjaub,

the Epidemic Cholera Morbus as it Visited the Territories subject to the Presidency of Bengal in the Years 1817, 1818, and 1819.' By James Jameson, Calcutta, 1820. (In German, Stuttgart, 1832.) W. Scott, 'Report on the Epidemic Cholera as it appeared in the Territories subject to the Presidency of Fort St. George, &c.,' Madras, 1824. 'Report on the Epidemic Cholera which has raged throughout Hindostan and the Peninsula of India since August, 1817,' Bombay, 1819.

there is no information as to the spread of cholera during the year 1819; the first accounts of severe cholera epidemics in those regions belong to 1820 and 1821, in which years also many other parts of India suffered from repeated visitations of the pestilence.

As early as the end of 1818, the disease had crossed the borders of its indigenous territory in the course of this pestilential progress. In December of that year it was at Jafnapatam, the most northern point of Ceylon, and thence it had travelled, during the early months of 1819, to Trincomalee, Kandy, and Colombo, and so ultimately over the whole island, carrying devastation with it. In the beginning of 1819, it had spread from Bengal to Nepaul and Arracan and thence over *Burmah* and *Siam*, and along the peninsula of *Malacca* to Singapore, the most southern point of Further India. At the same time (May) the disease appeared in Sumatra, and, in the beginning of the following year, in Java, Borneo, and others of the *Sunda Islands*, which continued to be severely ravaged by the pestilence during the three years following. From Ceylon, the disease was imported in 1819 into *Mauritius* and *Réunion* (then called Bourbon), and thence, in the beginning of 1820, to the *East Coast of Africa*, where it was limited, at this its first outbreak, to the Zanzibar coast. The *Moluccas* and *Phillipines* were reached in 1820, and in the same year the cholera appeared for the first time in the *Chinese Empire*, through which it spread devastation from one end to the other during the two years following, and crossed to Nagasaki (Japan) in 1822.

From India cholera began its travels westward somewhat later than its easterly and southerly progress. The first point visited by the sickness in that direction was Muscat, on the east coast of Arabia, whither it had been brought from Bombay in the spring of 1821; it spread quickly along the coast as far as the confines of *Mesopotamia*, crossed over to the coast of *Persia*, where it appeared first in Bender-Abassi, somewhat later in Bushire, travelled thence in a north-westerly direction along the coast towards the Euphrates, towards the north-east into the interior of Persia, and by caravan routes through Yezd into the north-eastern provinces of the country. From Busorrah, the first affected point in Mesopotamia, the epidemic spread along the Tigris to Bagdad, and along the Euphrates to the town of Anah situated on the confines of the Syrian Desert; from Bagdad it was carried by a Persian army in the autumn of 1821 into the north-western parts of Persia. The approach of the cold season brought the epidemic to an end over the whole of this Asiatic territory; but in the spring of 1822 it appeared afresh in most of the districts previously visited by it, and this time it pressed on along the Tigris to Mossul and *Kurdistan*, and westwards through Mardin, Diarbekir, and Urfa, to *Syria*, reaching Aleppo first in December, soon after which it died out. On Persian soil, too, the pestilence took a fresh start of considerable extent; in August it had reached to Tabriz and thence it penetrated into the provinces of Gilan and Mazenderan. The epidemic again subsided during the winter of 1822-23, to break out in a wider compass in the

spring of 1823; from Antakija (the ancient Antiocheia) and Ladakijeh (Laodicea ad mare), the points first attacked, the disease overran the coast of Syria as far south as Palastine. In another direction, it came on to Damascus, and in May it reached *Russian* territory from Persia, entering Transcaucasia along the Kur and travelling as far as Tiflis; in August it appeared in Baku, and was carried thence on shipboard to Astrakhan (22nd September); but it died out there as early as October on the severe cold setting in, and it was extinguished in like manner at all points of Central Asia that had been attacked up to this time, without recurring in the spring following.

Thus ended the first act of the cholera drama on extra-Indian soil. Within the period from 1817 to 1823 the disease had spread over a territory of nearly one hundred degrees of longitude (from Nagasaki in 147° E. to the coast of Syria in 52° E.), and upwards of sixty-seven degrees of latitude (from Bourbon in 21° S. to Astrakhan in 46° 21′ N.), and in its western course it had come close to the frontiers of Europe, but without crossing them. The winter of 1823-24 had brought with it the complete extinction of the plague throughout the whole territory of Central Asia that had been affected by it. For a space of four years, the disease was again confined within the country of its origin, to begin, on the expiry of that period, a new career towards the borders of Europe and of Northern Africa, and thereafter, with rapid flight, to make the circuit of the whole globe.

§ 100. Second Pandemic 1826-37.

The beginning of the second pandemic of cholera dates from the year 1826.

Cholera having in that year again attained a somewhat wide distribution in Bengal, and having advanced along the Ganges and its tributaries through the North-West Provinces,[1] started from two points to travel westwards. From Lahore it came in 1827 to *Cabul*, *Balkh*, and *Bokhara*[2] by the caravan routes; in 1828 it spread from Khiva to the Kirghese hordes, and from them, in August, 1829, to Orenburg, where it spread over the whole government, and did not die out until the winter of the following year. By the second route the pestilence entered Persia for the second time in 1829; it broke out in the autumn of that year in Teheran, the capital of the empire, which had hitherto escaped,

[1] Macnamara, 'History of Asiatic Cholera,' Lond., 1876, p. 77, *et seq.*

[2] Burnes, in 'Calcutta Med. Transact.,' vii, p. 459.

died out during the winter, but reappeared when the warm weather of spring set in, and then advanced along its former route to Astrakhan, where it showed itself in the middle of July. From that centre it spread rapidly along the shores of the Volga to Saratow and Kasan, along the Caspian coast and the shores of the Ural to Uralsk, and finally along the Caucasian frontier to the country of the Don Cosacks. Before the end of 1830 the pestilence had obtained an extensive footing on *Russian* soil. It had spread northwards to Viatka and Perm, north-west to Jaroslav, Tver, and Novgorod almost to the confines of the Petersburg province; from the Don it had extended to the Crimea and the banks of the Dnieper, and along the latter to Kiew, Podolia, and Volhynia. Even the cold winter of 1830-31 set no limits to its progress, and in the first months of 1831 it had got as far as the western governments of Minsk, Grodno, and Wilna (which had been spared on the previous occasion), the Baltic provinces, and finally Poland, where the military operations of the Russo-Polish war contributed materially to its diffusion. In June the disease broke out in St. Petersburg, almost simultaneously in Orel and Archangel, and in July in Finland, where, however, it obtained a comparatively slight hold.[1]

While cholera was penetrating by this route into the heart of Europe, it was at the same time spreading to Mesopotamia and Arabia by a road once trodden before; and from those countries it was carried in the spring of 1831 by bands of pilgrims, on the one hand, to Syria and Palestine, and, on the other hand, by way of Suez to Egypt. It appeared in Cairo in July, and spread along the Nile upwards as far as Thebes, and as far down as Alexandria, so that it overran the whole Delta. The same year it was carried to Tunis by pilgrims. It was not until several years later that cholera attained its wider distribution on the North Coast of Africa.

The pestilence reached *Germany* from Russia by three routes. After the cholera had broken out, as we have seen, in Poland with the invasion of the Russian troops in February, 1831, it spread from Brzesko along the main roads to Biala, Lublin, and Warsaw, appeared in Kalisch in the end of June, and crossed the Prussian frontier at that point. It overran the departments of Posen and Bromberg, penetrated into the province of Silesia, and followed the course of the Oder to Brandenburg and Pommerania. In the last-mentioned provinces it spread to a comparatively small extent except in a few of the larger towns (Stettin, Frankfurt-on-the-Oder, Küstrin, Potsdam, and Berlin), while the department of Stralsund, as well as the districts of Uckermünde and Prenzlau, remained entirely exempt. In the department of Cöslin, also, the disease (imported from Western Prussia) occurred only in isolated cases; and it is worthy of note that the area of the disease became narrower the farther west it went, this circumstance being especially noticeable in the territories west of the Elbe, such as Prussian Saxony, the town of Hamburg, the duchy of Holstein, and other

[1] On the epidemics of cholera in Finland see particularly Quist, 'Om Kolera i Helsingfors, 1871, &c.,' Helsingfors, 1872.

localities. Lüneburg was the only place affected in Hanover at this first outbreak of cholera, the Duchy of Brunswick escaped entirely, while Bremen was not reached (from the Hamburg side) until October, 1834. It was not until the years 1832 and 1833 that the disease entered the Rhine provinces (from Holland), and then only to a very limited extent. In several of the affected districts last mentioned, cholera reappeared in the two following years; but the ravages of the disease in the group of countries lying to the west of the Elbe did not come within sight of the enormous sacrifice of lives that the plague exacted in the eastern parts of North Germany, more especially along the Vistula, in the low country between that river and the Oder, and in the province of Posen. The second route by which cholera entered Germany from Russia was by way of Danzig, and without doubt it was brought by Russian ships of war. It spread thence by Elbing to Königsberg, and westwards over a small part of the department of Cöslin. Simultaneously with its appearance in Danzig the pestilence had crossed the frontier of East Prussia and overran the department of Gumbinnen. Lastly, by a third route, the disease came from Russia to *Austria*. As early as January, 1831, it had crossed the frontier of *Galicia* from Podolia, and by the end of July it had spread over the whole of that country. It reached *Hungary* in June, and travelled, on the one hand, into Lower Austria, and, on the other hand, into the Banat and Styria. From Lower Austria cholera spread to Moravia and to Upper Austria, while the infection reached Bohemia from Silesia. In most of the districts above mentioned, the amount of sickness in 1831 was small; the epidemic proper appeared in them first in the spring and summer of the following year, and for that occasion it was limited to those provinces of Austria that have been named, while the mountainous region of Styria, Carinthia, and the Tyrol, together with the whole South-west of Germany, enjoyed a complete exemption. A few weeks after the disease had shown itself in Galicia it broke out in *Moldavia* (having entered from Bessarabia), and extended its progress thence to *Walachia*, *Bulgaria* (July), and *Roumelia*. From *Galatz* it was taken by the sea route to *Constantinople*, where it appeared in July (1831), and it was conveyed thence in September to *Smyrna*, probably by an American frigate.

From Germany the infection was carried to *Great Britain* before the end of 1831. The disease appeared in Sunderland in October, having been imported by a vessel from Hamburg; an outbreak quickly followed in Newcastle and Gateshead, and by December it was across the Scottish border at Haddington, in the beginning of January, 1832, at Tranent, a fortnight later at Musselburgh, in the beginning of February at Edinburgh, and in March at Glasgow; from the latter it was conveyed in the middle of March to Belfast, to Dublin at the end of the month, and in April to Cork. Thus, in the course of the year it spread over a great part of Britain, following the commercial highways chiefly, and the coast routes and rivers, while the mountainous parts of the country were little visited by it, and the Scottish Highlands not at all.

The outbreak of cholera on *French* soil corresponds with the time of its general prevalence in England. In the middle of March (1832) it appeared almost simultaneously in Calais and Paris, and from each of those points it spread with so extraordinary rapidity that the greater part of Northern France was overrun within the months of April and May. By the middle of June the disease had reached the southern departments. It had held straight on as far as the department of the Indre; thereafter it appeared in the Gironde and the Bouches du Rhône, which had been hitherto quite exempt, but now became centres for the spread of the disease in the south. Out of the eighty-six departments of the country, there remained thirty-six completely exempt; they were mostly those situated in the southern and eastern parts of France, while, of the level country, only the Dordogne and the departments of Loiret, Sarthe, and Vienne escaped. In the spring of 1833 the disease broke out afresh in certain of the northern and north-eastern districts, but to a very limited extent. I shall afterwards speak of the outbreak of cholera in the south of France in 1834 which became so fatal to Southern Europe.

The pestilence was imported from France into *Belgium* in the beginning of May, 1832. Its first appearance in that country was at a village in the province of Hainault on the French frontier; shortly afterwards it was seen at Courtray (West Flanders), and it spread to Brussels by way of Ghent, to Luxemburg in the beginning of July, to Antwerp in the middle of that month, and so overran a large part of the country, although with moderate intensity. Cholera showed itself in the *Netherlands* at the time when it was epidemic on the French, Belgian, and English coasts. It appeared first in Scheveningen in the end of June, 1832, and at the Hague and Rotterdam in the middle of July; but it met with a check, and, notwithstanding a fresh outbreak in 1833, it attained a comparatively slight distribution. In true epidemic form it was observed only in the provinces of North Brabant, North Holland (especially at Amsterdam), South Holland (especially at the Hague), Friesland (at Leeuwarden), Gröningen (in the town of that name), and Drenthe (at Assen). Connected with this epidemic, as we have already seen, comes the outbreak of cholera in 1832 and 1833 in Rhenish Prussia, whither the disease was imported from Rotterdam to Emmerich; it was epidemic at Ruhrort and Mülheim, and at Duisburg, but in the neighbourhood of Aix it occurred only in isolated cases; so that this part also of Western Germany was only slightly disturbed by it.

Of Scandinavian countries, *Denmark* completely escaped the pandemic. In *Norway* the disease occurred in the autumn of 1832 only at Drammen and in its immediate neighbourhood. It became somewhat more widely spread in the following year (especially at Christiania, Ager, Holmestrand, and Moss); but it was not until August, 1834, that it reached the height of a severe epidemic (particularly at Frederikstaad, Moss, Soon, Krageröe, Farsund, Egersund, and other places). In the same year (1834) cholera first visited *Sweden*. It made its

appearance in Gottenburg, and spread thence along the river Götha, and onwards over a great part of the low inland plain, especially around the great lakes, and gradually northwards as far as the 60th parallel of latitude. Only Malmö, Kolmar, and Wimmerby of the southern districts entirely escaped the disease.

Before cholera had reached the South West of Europe it had already crossed the ocean to the *Western Hemisphere*. Carried by Irish emigrants, it reached *Canada* in the beginning of June, 1832, and spread with enormous rapidity along the St. Lawrence and its tributaries, as well as along the shores of Lake Ontario, covering a great part of Upper and Lower Canada, to Lake Champlain on the one side, and to Cornwall, Greenwich, and Bytown on the other. Almost at the same time it reached the *United States*, coming from Canada to Detroit (Michigan), and also directly to New York with emigrants from Europe. As early as the beginning of July it had spread along the east coast as far down as Philadelphia, in the middle of August we find it in Maryland and Virginia, and at the beginning of October in Kentucky, whence it followed the course of the Ohio and came to Indiana and Illinois. At its first outbreak the pestilence reached no great extent in the Western States, but it assumed far greater proportions in the year following. In October, 1832, cholera appeared at New Orleans and spread rapidly along the Mississippi over a part of the Southern States; it looked as if it had died out during the winter, but in the following summer it broke out there afresh. This time it spread with great violence through the Southern, Central, and Western States, invaded the Indian Territory, where it wrought devastation among the natives, and crossed the Rocky Mountains to the Pacific. The eastern countries of North America remained almost free from cholera during 1833, but in 1834 the disease reappeared in them, and on this occasion it extended as far as Halifax, Nova Scotia. In the year 1835 the disease was imported once more into New Orleans (from Cuba), but this time it appears to have been limited to that city and to Charleston, South Carolina. The cholera reached Mexico—it is said by importation from Cuba—in June, 1833, appearing first in Vera Cruz and Tampico, and spreading from the coast rapidly into the Tierra Fria to as great an altitude as 6-7000', and so upwards to the City of Mexico and to Puebla. *Cuba* was the only island of the *West Indies* visited by this pandemic, and particularly the towns of Matanzas and Havana, where the plague broke out as early as February. In 1835 there was a new outbreak, which spread over the whole of the south coast. In that year also the cholera showed itself for the first time on the continent of South America, but it remained confined to a mild epidemic on the coast of *Guiana*. Finally, the disease first reached *Central America* in 1837; we have no definite information about this epidemic except from the States of *Nicaragua* and *Guatemala*.

While cholera had thus been overrunning a great part of the Western Hemisphere, it had been at the same time penetrating into regions of Southern Europe that had hitherto escaped it. In January

of 1833 the disease obtained a firm footing in *Portugal*, having been carried, it was said, by an English ship to the harbour of Oporto. It appeared first in the small shipping place of St. João da Foy, and shortly after in Oporto itself, then in Coimbra, by the middle of February in Aveiro, and in Lisbon at the beginning of April. In the middle of January of the same year cholera appeared in *Spain*; the Galician seaport town of Vigo was its starting-point, and it then spread to Santiago, Calzada, and Pontevedra, upwards throughout the province of Galicia, and it thence turned southward and travelled without interruption down through Estremadura and Andalusia to the Mediterranean. A still more considerable extension of the disease took place in Spain in the following year. This time it included in its seizure those northern and eastern districts which had formerly escaped; and that epidemic may be looked upon as the starting-point of a new invasion of cholera over a great part of Europe. In December of 1834 cholera broke out in Marseilles, having been brought from a Catalonian port. It spread next to Cette, and to a few localities in the neighbourhood of that town, died out with the setting in of cold weather, reappeared in March, 1835, overran a great part of the South of France, which had almost escaped up to that time, and reached *Piedmont* in the summer of 1835. From Piedmont it spread in one direction from Nice along the Ligurian coast into Tuscany (particularly to Rossignano), as well as in a northeasterly direction over the Maritime Alps to the upper basin of the Po. Savoy, as well as Central and Southern Italy, remained exempt, and even Lombardy was little visited by the plague that year. On the other hand, it reached *Venetian* territory in the beginning of autumn; it had been imported from Piedmont to an Arriano island, situated in the Po, it then traversed the districts of Loreo and Treponti, reached Venice in the beginning of October, and proceeded along the coast through Palestrina, to Adria, coming in November to Padua, Vicenza, and Verona, and ultimately into Milanese territory (Bergamo). The cold of the approaching winter brought the pestilence to an end there, but in March, 1836, it reappeared in all the localities previously affected, and spread in the course of the year over almost the whole of *Italy*. It became general especially throughout Lombardy and Venetia, thereafter throughout part of the States of the Church, and ultimately in the Kingdom of Naples, where it occurred in a few places (Berletta, Bari, &c.) as early as July, and in the capital in August. From Neapolitan soil it arrived in *Sicily* in January, 1837. In that year also (1837), cholera appeared in *Malta* for the first time. From Como the disease made a slight incursion into *Switzerland*, visiting the canton of Ticino in July, 1836, but remaining confined to the districts of Mendrisio and Lugano. From Venetian soil, however, it proceeded towards the southern frontier of Austria, and, for the second time, came to be widely diffused over Austria and Germany. Starting from Roveredo, it broke out in the German Tyrol, which had hitherto remained free; and from the north coast of the Gulf of Venice it reached *Dalmatia*, appeared in Trieste in March, 1837, then spread through *Illyria* along

the main roads, especially through those parishes belonging to the Duchy of Carniola, made a small inroad into Styria, broke out in April in the Archduchy of Austria, and especially in Vienna, whence it extended eastwards to Hungary (but only to a moderate extent), and from there to Galicia, and northwards to Bohemia and Moravia. It was in this year that the South West of Germany became for the first time the scene of an epidemic of cholera, certainly a very limited one. In August the disease was imported into *Bavaria* from the Tyrol. It appeared first in the frontier village of Mittenwald on one of the highroads between the Tyrol and Munich, soon after in Alt-Oetting and Neu-Oetting, in October in Munich and a few villages in its vicinity; but it obtained on the whole a slight diffusion, and it died out in the beginning of December. From Galicia the cholera came to Poland, and thence along the Vistula into Western Prussia, and on the other side into Silesia; so that in the end of June it had reached the department of Marienwerder, Danzig in July, then, in an easterly direction, Königsberg (beginning of August) and Gumbinnen (middle of August), Breslau in the end of July, and Berlin a little later. But in all these places, with the exception of Berlin and a few other towns in North Germany, such as Brunswick, Stettin, and Hamburg, the epidemic was marked by a limited tendency to spread, and before the middle of autumn it was everywhere extinguished.

Spreading from India in an easterly direction, cholera had meanwhile in 1830 reached China for the second time, and Japan in 1831. At the same time it had found a new field for its extension on the continent of *Africa*. Besides *Egypt*, which we have already seen to have been attacked in 1831, the pestilence appeared in *Algiers* in the autumn of 1834, first in the port of Mers-el-Kebir (province of Oran), and extended thence along the strip of coast to Bona. Cholera was again disastrously prevalent there in the years 1835 and 1837. The outbreak in the latter year was probably the continuation of a pestilential progress from Egypt through Tripoli and Tunis, the wider ramifications of which may be seen in the epidemics that prevailed at the same time (1837) in *Abyssinia*, on the East Coast of Africa from *Somali-land* to *Zanzibar*, and in the Soudan countries, *Khartoum*, *Kordofan*, *Darfour*, and *Waday*.

The winter of 1837-38 brings the second pandemic of cholera to a close; the disease died out during the winter at every point in the immense territory which it had overrun in the period from 1826 to 1837. For the next ten years the soil of Europe, Africa, and America was completely free from it.

§ 101. Third Pandemic, 1846-63.

The third pandemic of cholera begins with the year 1846.

As early as 1840 and 1841, cholera had attained to an unusually wide extension on *Indian* soil, and had spread to *Further India*, the *Philippines* and *China*, to *Cabul* in 1842, and thence in the following year in a south-easterly direction to *Peshawur*, *Lahore*, and the *North-West Provinces*. In 1844 the pestilence broke out afresh in Cabul, it appeared at Herat in July of that year, at Samarkand and Bokhara in the following months, and towards the end of the year in the eastern provinces of *Persia*. There the disease lasted through the winter, and in the spring of 1846 it spread over the greater part of the country. In May it had already attacked Asterabad and Teheran, whence it proceeded in a north-easterly direction towards *Caucasia* and *Armenia*, in a southerly direction through Ispahan and Shiraz to the shores of the Persian Gulf, and westward towards *Mesopotamia*, where it first appeared at Bagdad, and thence travelled southwards to Bussorah, and northwards along the Tigris through Mossul as far as Diarbekir. From the Persian Gulf the disease reached the coast of *Arabia*, along the whole length of which it spread; while from *Transcaucasia* it arrived on the shores of the Caspian at the beginning of 1847. In those regions of Nearer Asia the epidemic lasted, not without interruptions, through the two following years (1847 and 1848), for which we have records of its prevalence in Persia, Mesopotamia, and Erzeroum. From the shores of the Caspian, the disease spread in the year 1847 northwards and westwards in three lines. In the basin of the Ural, where it joined hands with another invasion coming from Bokhara, it appeared first in spring in the government of *Orenburg*, and it overran a great part of *Siberia* with such rapidity that Tobolsk was reached previous to July. Proceeding in a westerly direction it reached the coast of the Black Sea in August, appeared in Trebizond in September, and in Constantinople in the end of October. It was not until March, 1848, that the cholera in that city attained to really epidemic sway, and then it spread over a great part of *Turkey*, the *Danubian Principalities*, *Hungary*, *Asia Minor*, *Syria*, and *Egypt*. In the last-named country it broke out as early as the month of April, and travelled through *Tripoli*, *Tunis*, and *Algiers*, as far as *Morocco*. In all of the countries here named, the disease raged in the two years following as well (1849 and 1850). To the same period belongs also a new outbreak of cholera in *India*, *Further India*, and the *Indian Archipelago*, as well as the appearance of the disease in two regions of Southern Europe hitherto spared: viz. *Greece*, where it first showed itself (in 1848) in the island of Schiathos, extending next year to other islands; and *Malta*, where it prevailed to a moderate extent in 1848, and with much greater intensity in 1850.

In the meantime, as early as the beginning of 1847, cholera had entered *European Russia* at two points. One of these was Astrakhan,

where it broke out in April and travelled along the Volga into the interior of the country; the other was the shore of the Sea of Azov, from which it spread upwards along the course of the Don, as well as westwards to Odessa. It now travelled with great rapidity in all directions, and by the autumn it had gone eastwards as far as the Ural, northwards to Archangel, and westwards to the Baltic. The winter of 1847-48 put an end to the epidemic, but it broke out anew in the spring of 1848, and again attained to an area of diffusion as large as the whole empire, including Poland. In November the cholera was completely extinguished in Russia, and in the two years following, when the disease was prevalent over the greater part of Europe, the Russian Empire remained free from it, except at St. Petersburg, where isolated cases continued to occur, and in Finland, where the disease was still epidemic in 1849, but only to a slight extent, just as it had been in 1848.

In the early summer of 1848 cholera came from Russia to *Germany*. It occurred first in Pommerania, Prussian Saxony, and the Brandenburg Mark, somewhat later in Hamburg, Bremen, Hanover, and Brunswick, and in the autumn in Posen, East Prussia, West Prussia, and Silesia. It survived the winter in several localities, broke out in them with increased violence in the spring of 1849, and now overran Rhenish Prussia, which had suffered but slightly in the first pandemic. In the following year, also (1850), the disease prevailed epidemically, although with less intensity, at numerous points in the north and west of Germany. The south and south-west of the country remained almost entirely exempt from this pandemic; here and there a few sporadic cases of cholera appeared, and in 1849 the disease attained a true epidemic character, but within narrow limits, in Mannheim and a few localities in the Jaxtthal and Enzthal (Würtemberg).

In the autumn of 1848, cholera appeared in *England* and *Scotland*. The first place to be attacked was Hull, whither the disease had been imported in the beginning of October by a vessel from Hamburg. It soon showed itself in Edinburgh, London, Gravesend, Plymouth, and Sunderland, later in Glasgow and Dumfries, and became widely distributed in that year throughout Great Britain and Ireland. In many of the affected districts it lasted through the winter, to overrun a great part of the British Isles with increased intensity in the spring of 1849. The end of this epidemic falls in the late autumn of 1849; and in 1850 the whole of Britain was free from cholera.

Almost at the same time as England, the *Netherlands* and *Belgium* were visited by the disease; and, in those countries also, the disease reached its height in 1849. In the Netherlands, where the pestilence broke out in October, the provinces that suffered most were North and South Holland, Utrecht, Groningen, Overyssel, and Gelderland (of 23,267 deaths from cholera in the whole kingdom, 20,498 occurred in that province alone). In Belgium, the provinces most visited were Brabant, East Flanders, Hennegau, and Liege, while the disease was much more limited in Limburg, Luxemburg, and Namur. In these regions also, the epidemic was quite extinguished by the end of 1849.

Of Scandinavian countries *Denmark* and *Norway* remained almost entirely exempt in this pandemic. In the former country, sporadic cases of cholera occurred in 1848 at Dragör (on Amagar), at Copenhagen, on Korsör and Bornholm, and in Alborg in 1850; while the disease prevailed in true epidemic form, but only to a small extent, at Bandholm (upon the island of Laaland) in the summer of 1850. In Norway, during the autumn of 1848, Bergen, and a few shipping places in the district of Bergenhuus as far down as Stavanger, and Christiania in 1850, were attacked; but these also suffered only slightly. It was in *Sweden* that the disease attained its widest distribution; in 1850 it spread from the south coast (Malmö and Gottenburg) over a large part of the country, but even here its intensity was comparatively slight.

In *Austria* and *France*, cholera came to an outbreak first in 1849; and, in both countries, it prevailed as an epidemic to the close of 1850 (in Bohemia, particularly in Prague, to the end of 1851). In Vienna and other places in Austria proper, it appeared, in the first months of 1849, and in Bohemia and Moravia in the month of May; it came to Carniola (only the southern part) in August, invading Istria (Trieste) at the same time. The outbreak of cholera in *Northern Italy* stands in connexion with this epidemic. The disease was brought by Austrian troops to Venice in the summer of 1849, and spread thence by way of Verona to Brescia and Peschiera. It is highly probable that the appearance of the disease in the *Canton of Ticino* (limited as before (1836) to the district of Mendrisio), in the summer of the same year, was also connected with the Austro-Italian outbreak. In *France* also, the cholera had shown itself as early as the first months of 1849. The seaport towns on the northern coast formed its starting point; by the month of February the disease had taken firm hold at various places in Normandy and Picardy, it appeared in Brittany in April, and during the next three months it spread continuously in all directions over the whole country.

With the exception of the *Iberian peninsula*, which remained entirely exempt from this epidemic, the disease had spread over the whole continent of Europe. Simultaneously, it had reached *North America*, arriving as early as 1848. In December of that year, cholera occurred at one and the same time in New York and in New Orleans, having been imported from Europe by emigrant ships. The same month it spread along the Mississippi to Memphis, and by the sea route to Texas; and, in the course of the year 1849, it overran the whole of the States lying to the east of the Rocky Mountains. In April of that year it was imported into Canada, in like manner by emigrants from Europe. The pestilence continued throughout the year 1850, being particularly widespread in the Western States, in the basins of the Mississippi and Ohio. In October, it came, by importation from Panama, to San Francisco for the first time; and from that centre the valleys of the San José, San Joaquim, and Sacramento were infected. North America was not free from cholera in the years 1851 and 1852 as well; we have records of its epidemic prevalence from New Orleans,

St. Louis, and Quebec for 1851, and from Buffalo, Chicago, St. Louis, and a few places in Ohio, Kentucky, and Iowa, for 1852; but it was not until 1854 that the disease again came to be generally prevalent, and then only in consequence of new importations from Europe.

The sickness came to *Mexico* in 1849 by two routes; the first from Texas to Matamoras and other seaports on the east coast, whence it travelled quickly into the interior, appearing in Monterey in the end of March, in Saltillo in April, and spreading thence southwards to Zacatecas, south-westwards to Durango, and north-westwards to Chihuahua; the second, in summer, from Panama to the west coast, where Mazatlan and Acapulco were principally affected. In the following year cholera was again epidemic at many points in Mexico, at Vera Cruz, Tampico, City of Mexico, Puebla, San Luis Potosi, and other places. Of *Central American* States, *Panama* was on this occasion the only one visited by the cholera; the disease came first to Chagres about the end of 1849 on board a ship from New Orleans, and travelled quickly to Panama along the overland route. Connected with this epidemic there is, on the one hand, as we have seen, the outbreak of cholera on the West Coast of Mexico, and on the other hand, its appearance on *South American* soil, to wit in New Granada. From Chagres, the disease reached Cartagena and Santa Marta by the great military road, and then ascended the Rio Magdalena as far up as the plateau of Santa Fé de Bogota. According to somewhat unreliable accounts, it penetrated also to *Ecuador* and was prevalent in Quito. All the rest of South America escaped this epidemic. Much more destructive was cholera in the *West Indies* continuously from 1850 to 1854. It appeared first in 1850 in Cuba and Jamaica, where it continued to cause frightful ravages for several years following; the year after (1851) it was in San Domingo, then in the Bahamas (1852), and in the two years following it visited Porto Rico, Nevis, Tortola, Grenada, St. Thomas, Barbadoes, Santa Lucia, Trinidad, and other islands. This epidemic died out at the end of 1854.

A remission from 1850 *to* 1852.—Meanwhile, in the Eastern Hemisphere, a remarkable remission of cholera had set in with the close of the year 1850. This applies particularly to the whole of the South and West of Europe, while in the Eastern and Northern parts of the continent, there were observed only scattered epidemic outbreaks of the disease—at a few places on the Baltic (especially in Pommerania), in Bomehia, in the adjoining districts of Prussian Silesia and in Poland. In Africa also the disease was almost completely stamped out; it prevailed to a small extent in Algiers and Morocco, and in summer it appeared (for the first time) in the *Canary Islands*, remaining limited to Grand Canary.

A new and severe visitation of cholera over a great part of the globe was preparing in the year 1852. In the spring of that year, the disease broke out afresh almost at the same moment in Persia and Mesopotamia, and in Poland, countries in which it had survived the winter. From Poland it spread first (during the summer) into the adjoining Russian Governments of Volhynia and Grodno on the one hand, and on the other hand into the Prussian provinces of Posen, Silesia, and West Prussia, gradually extending over part of East Prussia, Brandenburg, and Pommerania, where, however, it was confined within small circles. In autumn, the disease reached the Western Governments of Russia (Kowno, Minsk, Courland, Livonia, Esthonia, and as far as St. Petersburg). In the end of the year (November and December), it arrived in Transcaucasia from the Persian frontier. Thus there was formed in that year an extensive focus of infection over the East of Europe, out of which a new pandemic arose, destined not to come to an end until eight years after.

In *Russia*, the pestilence maintained itself over a larger or smaller area until the year 1862. It was most severe in 1853, 1855, and 1859; it was met with only in St. Petersburg and Cronstadt in 1860; in the two following years it was observed within a small compass in the capital of the empire, thereafter becoming extinct, but only to overrun the country anew in a short time in consequence of a fresh importation from the East. In *Germany*, it happened, as in all former epidemics of cholera, that the plain of North Germany (East and West Prussia, Posen, Pommerania, Brandenburg, Mecklenburg, Schleswig-Holstein, Brunswick, Saxony, Hanover, and a few places on the Lower Rhine) was chiefly attacked, and next to it Silesia. As in Russia, the disease was most severe in those regions during the years 1853, 1855, and 1859. It was all the more remarkable that there should have been an epidemic in the South-Eastern part of the country (Bavaria) in 1854, in which year North Germany was almost entirely free from cholera, while in the South-West of Germany the disease was observed on this occasion again in merely sporadic cases or in small epidemics (as in the Palatinate, at a few places in Baden and Würtemberg, at Darmstadt and at Frankfort-on-the-Main). This Bavarian epidemic, which issued from Munich and spread in a wide sweep over the whole country, was probably connected with the prevalence of the disease in Austria. That country was quite free from cholera in 1853; it was invaded in 1854, but only to a moderate extent, apparently from the Danubian Principalities; much more severely, however, in 1855, when the disease at the same time penetrated from Italy to Istria, Dalmatia, and the adjoining region; so that with the exception of Upper Austria, Styria, and Carinthia, all the countries of the empire (including Hungary) suffered severely in this epidemic. But in Austria the disease died out completely with the close of the year 1855, while in Germany, as we have seen, it was not until the year 1859 that it reached its limit.

The first severe epidemics of cholera that occurred in *Scandinavian* countries, in *Finland*, *Sweden*, *Norway*, and *Denmark*, stand in con-

nexion with the general European outbreak in the year 1853. In *Sweden* the disease spread from Ystadt and Malmö progressively northwards as far as Umeå, and it was not until 1859 that the pestilence came to an end, having recurred year after year. Next to 1853, the year of greatest severity in Sweden was 1857; the chief centres in this epidemic, as in the previous ones, were the basins of Lake Malar and Lake Wetter, the valley of the Göta-Elf, and the southern belt of coast from Malmö round to Karlshamn. In *Norway* the area of the disease in 1853 extended from Christiania and other points on the coast to far inland, and included especially the districts of Smaalenene, Akershus, Jarlsberg, and Laurvig, with certain parts of Buskerud and Bratsberg. With the exception of slight epidemics in 1855 at Christiania and Tonsberg, and at Bergen in 1857, Norway remained quite free from cholera in subsequent years. In *Denmark* the disease spread in 1853 from Copenhagen over almost the whole of the island kingdom; here, too, it occurred subsequently to that year on two occasions only, in 1857 at Copenhagen and Korsör, and in Aarhus in 1859, both times to a limited extent. Cholera reached *Great Britain* in the summer of 1853, having been imported from Germany to a number of seaports, including Gateshead and Newcastle, Shields, London, and Liverpool; somewhat later it occurred also in Manchester, Edinburgh, Glasgow, and Dundee, lasted in those places throughout the following winter, and attained a wide distribution over the whole of Britain the year after. The localities that suffered most were London, Middlesex, Kent, Essex, Cambridgeshire, Durham, Oxfordshire, and South Wales. With the close of the year 1854, the disease died out at all points, and the whole kingdom remained free from cholera in the years following, with the exception of a small outbreak at Wick in 1859. In the *Netherlands*, where the disease appeared likewise in the summer of 1853, first in Rotterdam, Schiedam, and Dordrecht, it reached a considerable epidemic diffusion in that and the two following years, and again in 1859; in 1856 the cases were merely sporadic, and in 1857 and 1858 the country was free from cholera. The provinces of South Holland, North Holland, and Utrecht suffered most in these epidemics, and next to them, North Brabant and Groningen; while Drenthe, Overyssel, and Gelderland were less affected, and Limburg remained quite free year after year. Cholera arrived in *Belgium* first in 1854, and spread especially in the provinces of East Flanders, Brabant, Antwerp, and Liege; it died out entirely at the end of the year, and it was not until 1859 that it again reached an epidemic prevalence of considerable extent in the provinces of Antwerp, East and West Flanders, Brabant, and Hennegau.

In *France* cholera had already obtained a firm hold towards the end of 1853, first in Havre, and afterwards in Paris and elsewhere; but the disease was epidemic only within narrow limits. It was in the spring of the year following that it reached its general diffusion, extending continuously over almost the whole country, having started from three chief centres: an eastern one in the departments of the Marne

and Haute-Marne, a western in the departments of Vendee and Deux Sèvres, and a southern in the Bouches du Rhône. The southern provinces suffered most; that was the case also next year (in which the east of France, especially Alsace and Lorraine, had likewise a severe visitation); and even in 1856, isolated epidemics still occurred in Marseilles, Toulon, and some other places in the south, after which the disease came to an end in France. On this French epidemic of 1855, the outbreak of the disease in *Switzerland* was dependent, at least in part. In 1854 it was already prevalent at some places in the Canton Ticino, again imported from Italy; it had also come to the town of Aarau, its route thither being untraced. In the following year, however, it took a wider range; imported from France, it broke out first in Bâsle and afterwards in Geneva, and almost at the same time in Zurich; while it reappeared in Canton Ticino. It seems to have been introduced in 1854 from France into *Italy* also. It occurred first in Genoa and spread through Savoy, Lombardy, and Venetia, and along the Ligurian coast to Central and Southern Italy, as well as to Sicily, where Messina, Palermo, and Catania were visited with especial severity. The pestilence raged in Italy with much greater violence in the following year; only a few considerable districts in the whole peninsula (with Sicily) escaped it, and it was not till the end of 1856, in which year Northern and Central Italy suffered most, that the epidemic ended.

Spain formed one of the chief seats of the cholera epidemic of the years 1853-60. Introduced a second time through the port of Vigo, the disease broke out in that country in the summer of 1853, and spread over a great part of the province of Pontevedra (Galicia). In the spring of next year it was imported into Barcelona from Marseilles, and quickly appeared in Gerona, Tarragona, and other places in the Catalonian province, thereafter in Andalusia (Cadiz, Seville, Cordova, Malaga, Almeria), in Murcia, and in the interior, extending to Badajoz (Estremadura) and Madrid. In 1855 there was scarcely a province of the country remaining free from the disease; and in 1856 it still prevailed at numerous points, and especially in Andalusia. A very considerable remission seems to have occurred in the next two years; but in 1859 the disease broke out anew over a wide area, especially on the Eastern and Southern coasts, and in the following year it spread again over a large part of the country. Thereafter it ceased entirely. The diffusion of cholera over almost the whole of *Portugal* (from Oporto to Faro) coincided with the severe Spanish epidemic of 1855; the next year, too, the disease prevailed widely in Portugal, but it appears to have come to an end with the close of that year.

Russia and France were the points of departure for the epidemic in the South-East of Europe. In 1853 the disease penetrated from the former into *Transylvania* and the *Danubian Principalities*, while it was imported the year after by French ships of war into *Turkey*, spreading in that and the following year over the whole of Turkish territory as well as over *Asia Minor* and *Greece*. The cholera in Greece broke out at the Piræus in the beginning of summer, 1854, it appeared in Athens

shortly after, and in the islands of the Aegean; in 1855 the disease was still more extensively prevalent, and the Ionian Islands were also attacked. It appears to have died out completely in the winter of 1855-6 throughout this region, with the exception of Asia Minor, where there were still epidemic outbreaks in 1856.

On the continent of Asia, and in the Archipelagoes belonging to it, cholera obtained a not less extensive footing within the period that we are now considering. Starting from *India* (where it was widely epidemic, especially in 1852, 1858, 1860, and more particularly in 1861), it penetrated in 1852 to Java, Banda, and Sumatra, and spread next year over a large part of the *East Indies*. It appeared there anew in 1858 (especially in Celebes), and at the same time broke out in the Philippines. *Japan* was visited by severe epidemics of cholera in 1854 and 1857-59; within the same period fall the destructive outbreaks of the disease in *China* (where it continued prevalent till 1860), and in the peninsula of *Corea*, the southern and western regions of which received the epidemic from the capital. Severe epidemics of cholera overran Persia in 1853, 1856, 1857, and 1859-61, and in the latter years the disease occurred with equal virulence in *Afghanistan*, *Khiva*, *Turkestan*, *Mesopotamia*, and *Syria*. With the close of 1861, the disease appears to have been extinguished throughout that territory, Persia, at least, remaining entirely free from cholera during the next three years. Finally, there are for *Arabia* records of severe cholera epidemics in 1854 and 1855, in 1858 and 1859, and in 1862; the west coast seems to have been always the most severely visited, while the interior was invaded first in 1854 and once again in 1862.

During this period, cholera appeared on African soil first in *Algiers* in 1853, and extended to *Morocco* in the same year. But a much more important pestilential progress was that which began in 1855 and spread, in that and the following year, from *Egypt* to *Nubia*, and through *Tripoli*, *Tunis*, and *Algiers*, to *Morocco*. In the same period, the disease appeared in *Abyssinia* (for the second time), but only to a slight extent. It occurred also at Fogo in the *Cape Verd Islands*, having been imported from Italy, and in *Madeira*, introduced from Portugal; in both of which it was epidemic the next year as well. A new epoch of the disease begins in those (African) regions with the year 1858. In the first place, the cholera broke out afresh in Abyssinia, and to a greater extent than in the previous (second) epidemic; it appeared in 1859 in *Somali Land*, travelled along the coast to the Portuguese settlements in Mozambique, and broke out for the first time on the west coast of *Madagascar* and on the *Comoro Islands*. In that and the following year, *Algiers* was attacked anew, and the pestilence once more entered Morocco, where the coast towns of Tangiers, Tetuân, and Ceuta suffered most. Cholera was four times epidemic in *Mauritius* during this period, in 1854, 1856, 1859, and 1861-62, but only once (1859) in *Réunion*.

Lastly, in the *Western Hemisphere*, the renewal of the cholera pandemic is connected with importation of the disease partly from Europe, partly from the West Indies. In North America, how-

ever, it was almost limited to the year 1854, during which its area became nearly co-extensive with the continent. By the end of 1853, scattered epidemics had been developed in *Canada*, at a few points on the eastern seaboard of the *United States*, and at New Orleans and certain shipping places in the lower basin of the Mississippi. But it was in the spring of 1854 that the disease began to spread in all directions, and with such rapidity, that within a few months, the whole interior of the country (more especially the basins of the Mississippi, Missouri, and Ohio), the districts bordering the great lakes, both on the Canadian and the United States shore, and the whole of the seaboard on the east and south, from Montreal to Texas, were overrun by it. In Mexico, however, only the capital and a few places on the coast suffered. In 1855, there were still some scattered epidemic outbreaks on the continent of North America; after which it appears to have been entirely free for the rest of this pandemic period. In *Central America*, the disease broke out in 1856, first overrunning *Nicaragua* from the Atlantic to the Pacific, next appearing in *Costa Rica*—it is said for the first time—and extending in the following year to *Guatemala*, *San Salvador*, and *Honduras*. Meanwhile cholera had taken firm hold on South American soil. It was prevalent in *New Granada* in 1854, and in *Venezuela* in 1855. In the same year it showed itself for the first time in *Brazil*, at three points almost simultaneously—Para in the middle of May, Villa da Imperatriz and province of Amazonas in the beginning of June, and Bahia at the end of June. Shortly afterwards Rio de Janeiro was attacked, and in a few months the disease, travelling along the coast and up the Amazon and its tributaries, had covered an area which included the provinces of Bahia, Pernambuco, Sergipe, Parahiba, Rio Grande do Sul, Espirito Santo, and San Paulo. In November the disease died out at all points, and remained absent from the country in 1856 and 1857, although it was still prevalent in *Guiana*. In 1858, an epidemic again broke out in the Brazilian capital; after that, the disease showed itself in epidemic form in 1862 at several ports (Pernambuco, Rio, &c.), and in 1863 it was prevalent at Maroim in the province of Sergipe; but in none of those years was it so widely spread as before, and with the end of the last-mentioned epidemic, cholera remained absent from Brazil for many years.

The third pandemic of cholera embraces, therefore, a period of some fifteen years, during which the disease had spread over the whole Northern Hemisphere, and to 25° S. in the Old World, and to 30° S. in the New. Regarded as a whole, this pandemic shows numerous fluctuations of intensity, the maxima falling in the years 1849-50 and 1853-55. In none of the intervening years was the disease altogether extinguished on extra-Indian soil, and there is no reason for attributing the fresh outbreak of 1853 in Europe, Africa, and America, to a new importation of the morbid poison

from its native habitat. All the facts tell rather in favour of a continuous reproduction of the poison in extra-Indian countries, and that power of reproduction was exhausted only after the lapse of more than ten years.

§ 102. Fourth Pandemic—1865-75.

The fourth pandemic of cholera, and the latest hitherto, began in 1863, and it came to an end in 1875; it thus embraces, like the preceding one, a period of more than ten years. But it is distinguished from the previous pandemic, and in fact from all former cholera pandemics, by the peculiarity of the course that it took towards the West, and by the speed with which it travelled from Asia to the soil of Europe. Hitherto the disease had always penetrated to the continent of Europe through Afghanistan, Persia, and Asiatic Russia, and had taken more than a year to reach the European frontier by that overland route; but this time it took only a few days to reach Europe by sea from the coast of Arabia, and only a few weeks to overrun a large part of Southern Europe.

The starting-point of the sickness was the lower basin of the Ganges, where a severe epidemic of cholera had developed in 1863. In that and the two following years the disease visited the whole Presidency of Bengal, the North-West Provinces, the Presidency of Bombay, the central and southern parts of the Deccan, and Ceylon. Connected probably with that outbreak there was a disastrous epidemic in the East Indies in 1863 and 1864, and in China and Japan in 1864 and 1865. About the end of 1864 or beginning of 1865, the disease was brought on board ship from the Bombay coast to Hadramaut (south coast of Arabia), to the Yemen territory (southern part of the west coast of Arabia), and to Somali Land (east coast of Africa). In the first days of May it broke out in Mecca among the faithful assembled there to celebrate the festival, having been introduced by Indian pilgrims who had received the infection at some one of the points before mentioned. The outbreak of the disease among them was the signal for a panic, which was succeeded by a general flight, and thus the cholera was speedily carried in all directions along the tracks of the homeward-bound pilgrims. For the West, misfortunes began with the introduction of the disease into Suez, where a vessel arrived on the 19th of May from Jeddah with pilgrims belonging to Egypt and other North-African countries, among whom were cases of cholera. By this means Lower Egypt became infected in the beginning of June, and during

the weeks following the disease was carried thence by ships (and in fact partly in direct connexion with the transport of returning pilgrims), to Malta, Marseilles, Constantinople, Ancona, Valencia, and other places. It was carried, in like manner, to Asia Minor and Central Asia by pilgrims returning to their homes overland. Thus it was that the North of Africa and the South of Europe became the first seats of an epidemic of cholera which in that year (1865) and the two following overran a great part of both continents and extended even to the Western Hemisphere.

The first point in *Europe* affected by the disease was *Malta*, where it broke out on the 20th of June, and was carried to *Gozzo;* the epidemic died out in November, did not revive the next year, but occurred to a very limited extent in July, 1867, having been re-introduced from Italy. Marseilles was infected at the same time as Malta, by a ship with Algerine pilgrims returning home. The beginning of the epidemic there dates from the middle of June, and it came to a close in the end of December. From Marseilles the cholera spread quickly over the South of *France;* in July it was already prevalent at Toulon and Montpellier, in August at Arles, Avignon, and other places, in September at Aix, Cette, and Bordeaux, and simultaneously at Paris and Amiens; in October it was in the northern departments, and in November in the Vosges. Thus it had overrun the larger part of the country before the end of the year. In by far the most of the localities it was limited to a few cases, so that the total mortality from cholera in France that year was only 10,584. At many of the affected points the disease continued through the winter, and in the spring of 1866 it again reached the height of an epidemic, which prevailed especially in the north of the country, and most widely in the departments of Morbihan, Côtes du Nord, Finisterre, Meurthe, Seine-et-Marne, Calvados, Seine (Paris), and Eure. Next in order came the western seaboard (Charente-inférieure), and it did not spare the south of the country (Bordeaux, Marseilles, Ciotat). In the summer of 1867 cholera was still prevalent in a few of the localities formerly affected, especially those in the northern districts, but in epidemic form it was confined within narrow limits. In the autumn it died out everywhere, and from that time to the year 1873 France was quite free from cholera.

The disease reached *Italy* shortly after it came to Marseilles (beginning of July). The first place attacked was Ancona, whither the disease was brought by a ship from Alexandria, and it spread with great rapidity over almost the whole peninsula. The number of persons carried off by the pestilence was estimated at 13,000, Apulia suffering most (with 4617 deaths), and next to it Campania (with 3635) and the Marches (with 3429). In Italy, as in France, the disease lasted through the winter, and did not die out until March, 1866. It reappeared in August, and it now affected especially the regions of Campania, Sicily, Piedmont, and Liguria. In 1867, having again survived the winter at many places, especially in Sicily, it grew to a disastrous epidemic which carried off 130,000 victims. It was severest in Lombardy, Piedmont, Campania, the Abruzzi, and the Aemilia; but no province

was spared, and even the island of Sardinia had a visitation of it. In November the disease was extinguished at all points except Reggio (Calabria) and Messina, where it ceased in the course of the winter. Since that time (1867-68) Italy has remained free from cholera.

In *Spain*, the first place to be affected by cholera was Valencia, whither it had been brought, as was proved, by a traveller who had arrived from Alexandria viâ Marseilles. The outbreak of the epidemic dates from the middle of July, and almost at the same time the disease appeared in Gibraltar, imported without doubt by English troops from Malta; a little later it was at Cartagena, in August at Barcelona and Seville, shortly after at Toledo, Murcia, and Alcira, in September at Tortosa and Madrid, in October at Fuente de Cantos and other places in the province of Badajoz (on the Portuguese frontier), and in December at Santander (Old Castile). So that, with the exception of Galicia, Asturia, and the Basque provinces, no part of the country escaped the sickness altogether. Towards the end of the year the epidemic died out at all points, and, with the exception of a few small and isolated outbreaks in the following year, Spain has not since been visited by cholera. From Badajoz the disease crossed the frontier into *Portugal*, but as an epidemic it was restricted to the place first attacked (Elvas in the province of Alentejo). It came thence to Oporto, and in December it again crossed the Spanish frontier at Freixo d'Espadacinta (on the Douro); but at these two places there were only sporadic cases. Thus Portugal enjoyed an almost absolute immunity from this epidemic, and it has remained free from cholera in subsequent years.

For the East and North of Europe *Turkey* formed the point of departure of this epidemic. The disease appeared in Constantinople in June, 1865, having been imported, as we have seen, by a vessel from Alexandria. It spread thence in a northern and eastern direction over a great part of the Ottoman Empire, both along the west coast of the Black Sea to the mouths of the Danube, and along the banks of the Danube through Bulgaria, and also into the interior, special mention being made of severe epidemics at Phillipopolis, Seres, Varna, Kustendje, Rustschuk, Tultcha, and Widdin, as well as along the southern shore of the Black Sea to Sinope, Samsoon, and Trebizond. From Trebizond it penetrated (in the end of August) on the one hand into *Armenia* (Kars, Erzeroum, &c.), and on the other hand into *Caucasia*, thus reaching Russian territory, which had been already invaded from the west a few weeks earlier (*vide infra*). In the following year the cholera in Turkey appears to have been confined mostly to the Danubian provinces; in October and November it appeared at a few places on the Bosphorus without reaching a great epidemic extension. Next year it was limited absolutely to the northern part of Albania, whither it had penetrated in the spring of the year from Montenegro. During the four years following Turkey was free from cholera.

The country first infected from Turkey was *Roumania*. The disease appeared there in the beginning of August, 1865, at several shipping places (Ibrail, Galatz, Ismail, and Reni), and extended during the

months following through Moldavia and the eastern part of Walachia. It did not, however, attain to any considerable extent, and the whole mortality from it amounted only to four or five thousand, of which more than one half were in the district of Ibrail alone, about 1800 in Jassy, and about 1100 in Galatz. During the winter only sporadic cases were observed, but in May, 1866, the disease broke out in a new epidemic, again attacking the central and southern districts of Walachia, and carrying off more than 26,000 victims. During the same time the pestilence had been prevalent in *Montenegro*, and, although Roumania was free from cholera during subsequent years down to 1872, Montenegro was again affected in 1867, and gave the disease to *Herzegovina*, and, as we have already seen, to Albania.

The outbreak of the disease in Southern *Russia* stands in connexion with the Turkish and Roumanian epidemic of 1865. The places in that country to be first affected were Balta a village in the Government of Podolia, where the disease showed itself in the beginning of August among the members of a family newly arrived from Galatz and Odessa, to which latter it was brought by a vessel from Constantinople. The pestilence spread, but not in a fatal form, through the Governments of Volhynia, Ekaterinoslav, Kiev, Kharkov, Koursk, and Voronej, and became extinct towards the end of the April following, having caused a mortality of some 4000. While cholera was entering Russia on that side, it had penetrated, as we have already seen, into Caucasia, and had passed through Poti, Kutais, and Tiflis, as far as Elizabethpol and Erivan. It survived the winter in this region, as well as in the Governments of Southern Russia already mentioned; it reappeared at both points in the following summer (1866), but on this occasion it was not confined to the southern part of the empire, but overran the central, western, and southern Governments as well. At the end of June the disease appeared in St. Petersburg, Moscow, and Grodno; at the beginning of July in Tchernigov, Warsaw, Tver, Olonetz, and Orel; by the middle of that month in Chursk, Jaroslav, Esthonia, Kasan, Vologda, Livonia, Tambov, Kalouga, and Wilna; at the end of July in Pskov and Voronej; in August at Kowno, Nishni-Novgorod, Courland, Simbirsk, Mohilev, Astrakhan, Samara, Penza, Riazan, Vladimir, Perm, and Viatka; and lastly, in September, at Smolensk, Orenburg, and Ufa. The number of fatal cases in the whole empire was estimated at 90,000, of which nearly 18,000 belonged to Poland alone; the localities that suffered most were the Governments of Bessarabia, Podolia, Grodno, St. Petersburg, and Kherson. The disease again survived the winter, and broke out in the spring of 1867 as a fresh epidemic, which attained only to small dimensions, except in Poland, where it was as severe this year as last, the number of fatal cases amounting to 11,265. Towards the end of the year the pestilence had died out completely throughout the Russian empire, and it showed itself next year (1868) in epidemic form at one point only (Lipowitz in the Government of Kiev).

The other states of Europe in the year 1865 were either free from cholera altogether, or visited by it only to a small extent. In *Austria*,

where the disease had that year been prevalent at Fiume and Trieste, but only within a very narrow compass, it rose in 1866 to a very severe epidemic. It appears to have entered from Bukowina (where the disease appeared at the end of May), and it overran the whole empire including Hungary. The disease was felt most in the provinces that were directly concerned in the war of that year, including Lower Austria, with 10,000 deaths from cholera, Bohemia with over 30,000, Moravia with nearly 50,000, and Hungary with about 30,000. Neither did the disease spare Austria in 1867; but on this occasion it was mainly confined to Hungary (where it broke out as early as April), to Galicia, which suffered most in summer, and to Dalmatia and the surrounding country. The total mortality throughout the empire (excepting Hungary) amounted to nearly 32,000. Winter brought the epidemic to an end, and Austria was free from cholera during the next four years.

In *Germany*, the disease in 1865 showed itself, as in Austria, at only one point, and there it was very limited in its extent. It was imported into Altenburg (Saxony) by a family from Odessa; at the end of August it came to be feebly epidemic there, and in the following months it appeared at several towns in the Kingdom of Saxony (Werdau, Crimmitzschau, Zwickau, Glauchau, and others); but at only a few points was it epidemic, and the total mortality did not amount to more than 468. But next year (1866), Germany was visited by the pestilence much more severely. The Rhine country and Westphalia were the earliest attacked, the disease having been imported into them from Luxemburg (*vide infra*); the epidemic did not die out there until the beginning of the next year. In North Germany, the disease made its appearance in May, first in several seaports of Pommerania (Swinemünde, Stettin, Cammin, and others); in June it broke out at Hamburg and Berlin, and in the provinces of Prussia, Posen, Silesia, and the Kingdom of Saxony; in July it was in Prussian Saxony and in Brunswick; in August in Mecklenburg-Schwerin and Oldenburg and in September in the Duchy of Anhalt; in all which countries it came to an end at the close of the year. The disease during this epidemic carried off 114,683 persons in the Kingdom of Prussia. There also, the state of war helped materially to enlarge the area and to increase the intensity of the disease. The same is true of South Germany, where the Bavarian districts of Lower Franconia, Aschaffenburg, Swabia, and Neuenburg suffered; smaller epidemic centres formed also at a few localities in Hesse Darmstadt, and there was a considerable epidemic at Mainz, and some appearances of an epidemic in one district of Würtemberg (Utzmemmingen). But, for the rest, South Germany again enjoyed comparative immunity especially in its western parts, the cases of sickness everywhere remaining isolated, notwithstanding the importation of the disease into Speyer (from Belgium) and other places in the Palatinate, as well as into Frankfort and several places in Baden. In Rhenish Prussia, the cholera, as we have seen, survived the winter, and it prevailed with especial fatality at Cologne, Düsseldorf, Barmen, and Elberfeld during the following year; so that the mortality from

cholera in this one province amounted to almost the half (3418) of all that perished by it in the whole kingdom. The disease appeared in Upper Silesia in April, in the province of Prussia and in the governmental districts of Breslau and Bromberg in June, and in Mecklenburg-Schwerin in August; but nowhere did it amount to much. In like manner, in South Germany, where cholera appeared that year in several localities (Friesenheim and Mandach in the Palatinate, and a few places in Hesse-Darmstadt and Baden), the cases were for the most part only sporadic. Towards the end of the year, the disease died out everywhere, and next year (1868) it reappeared only at Essen, where thirty-eight persons died of it. In the years 1869 and 1870, Germany was quite free from cholera.

Among the larger territories invaded as early as 1865, we have to include *Great Britain*, where the disease was limited, however, that year to a few cases at Southampton and at Theydon Bois in Essex. The source of the disease was traced beyond doubt to a vessel which arrived at Southampton from Alexandria in the end of August. In May of next year, cholera appeared at Liverpool, having been imported from Rotterdam; it reappeared at Southampton in June, and broke out simultaneously in London, Llanelly (South Wales), South Shields, Newcastle, and other ports, and subsequently in Sheffield, Hull, Dublin, Belfast, and Manchester; in September it came to Edinburgh, Musselburgh, Dundee, and other places in Scotland. Thus it gradually overran the whole of Britain, so that no county or province of the three kingdoms was altogether spared, although, in by far the larger number of localities, the disease was confined to sporadic cases. In England the total mortality amounted to 14,378, of which 5596 belonged to London, 2600 to Lancashire, and 2033 to South Wales; for all Scotland the number was 1170; for Ireland 2501, of which 1459 were in Dublin alone. Next year (1867) the cholera in Britain occurred sporadically at a few points (Port Glasgow on the Clyde, and a few spots in the neighbourhood of Durham and Belfast), and there was a small epidemic outbreak in Jersey. Since that time to the year 1873 it did not return.

In *Belgium*, cholera had taken a firm hold as early as 1865, having broken out in Luxemburg in September of that year; the disease grew there to a considerable epidemic in November, reached its acme in March, 1866, and then died out somewhat quickly. In May, 1866, it reached Antwerp, having been introduced by emigrants from Germany, and about the same time it came to Brussels and Ghent; in June it appeared at Liege, Namur, Malines, Mons, Bruges, and other places, and in the end there was no province that it did not invade. The close of the epidemic dates from the end of November. It had caused a total mortality of 32,812, the provinces most severely visited having been Brabant with 9034 fatal cases, Liege with 5822, East Flanders with 5114, Antwerp with 4588, and Hennegau with 4104. In 1867 the disease was again observed in several parts of the country, but only in sporadic cases. In the years from 1868 to 1872, Belgium was entirely free from cholera.

During this pandemic, cholera entered the *Netherlands* first in 1866. It appeared in March at Rotterdam, in April at Utrecht and Delft, in May at Amsterdam, Alkmaar, Haarlem, the Hague, Leyden, and Dort, in June at Overyssel, Drenthe, and Groningen, and in July at Limburg. The number of deaths in the whole kingdom came to nearly 20,000, North and South Holland, Utrecht, and Groningen having suffered most. Next year (1867) the disease reappeared in epidemic form at a few places in the Netherlands (Rotterdam, Delft, Dort), but always within a small compass; South Holland was again the greatest sufferer, but even there the number of fatal cases was only 1033. From 1868 to 1872 the country remained free from cholera.

Of *Scandinavian* countries, *Denmark* was unaffected by the disease except to the extent of four cases in Copenhagen; in *Norway* the disease was observed in August, 1866, at Christiania and a few places on the coast, but mostly in sporadic cases only; in *Finland* also there were a few scattered cases during the years 1866-68. It was only in *Sweden* that the disease prevailed in 1866 to any considerable extent, although not with great severity; it broke out in June almost simultaneously at Gottenburg and Stockholm, somewhat later at Gefle, and from these points it overran the greater part of the country. The epidemic carried off in all 4503 persons, of whom 1359 were in Gottenburg and Bohuslän, 656 in Stockholm, and 654 in Easter Gothland.

Finally, we come to the outbreak of cholera in *Switzerland* in 1867. On this occasion the Canton of Ticino was again infected, from Lombardy, in the month of July; and about the same time a small epidemic developed in Zurich in consequence of an importation of the disease by a family from Rome; there were, besides, a few scattered cases observed in the Valais, and (on not quite reliable evidence) in Lausanne and Solothurn. By the month of October the disease was everywhere extinct, and since that time it has not shown itself in Switzerland.

In the *Western Hemisphere* the first seat of the epidemic was *Guadeloupe*, where the disease appeared in the end of October, 1865, and raged with great fatality (causing nearly 12,000 deaths) till April of the following year. The origin of the disease there is veiled in considerable obscurity; this much only is certain, that, a few days before the outbreak, two French ships from infected ports, the one from Marseilles and the other from Bordeaux, arrived at Pointe-à-Pitre, and that the first cases of sickness occurred among such of the inhabitants as had come into direct contact with the vessel from Bordeaux, on board which there had been one death during the voyage, from a cause not specially stated. Soon after the outbreak on Guadeloupe, isolated cases of cholera occurred on Martinique and Dominica. In November of the following year (1866) the epidemic broke out on *San Domingo*, and in the spring of 1867 at Havana and other places in *Cuba*, where it continued its ravages for three years; in March, 1868, it was in *St. Thomas*. Further information about the extension of the epidemic in the West Indies is wanting, but cholera does not appear to have recurred there after 1870.

The mainland of *North America* remained free from the disease during 1865, with the exception of a series of cases of obscure origin which occurred in November and December in the emigrants' hospital on Ward Island. It was in 1866 that the disease first took firm hold at three points—Halifax (Nova Scotia), New York, and New Orleans; and it was from the last of these that the disease spread most widely over the *United States*. The importation into *Nova Scotia* was through a vessel from Liverpool bound to New York, which was obliged to make for Halifax owing to the outbreak of cholera among the passengers and crew. The number of cases among the residents in the port was but small, and in all the cases a direct or indirect communication with the vessels could be traced. The disease gained ground no farther in Nova Scotia, and *Canada* also remained exempt, with the exception of a small epidemic at Stratford, Canada West.

In *New York* the disease broke out on the 1st of May, twelve days after the arrival of the above-mentioned vessel from Halifax; but no definite connexion could be traced between the first cases of sickness and the ship or her crew and passengers. In Brooklyn and on Governor's Island, cases first occurred in July, at Philadelphia and other places in *Pennsylvania* shortly after, and subsequently at Baltimore and a few other points on the East Coast. But of those places, it was only New York in which cholera spread at all widely, the number of fatal cases there from the 1st of May to the 24th of November having been 1210. From New Orleans the disease took a much wider range, travelling along the Mississippi basin and its tributaries as far as Illinois and Iowa. Next to New Orleans, the towns that suffered most were Memphis, Nashville, and St. Louis. The disease occurred sporadically also on the south coast (especially in Mobile), in a few ports of Texas, and on the Atlantic coast, particularly in Savannah, whither it was brought by sea with troops from New York. This epidemic reached its height in August, declined towards the end of the year, but revived the year after (1867), when it prevailed mostly in the Western States, in Missouri, Kansas, Arkansas, and the Indian Territory, and in Texas especially along the banks of the Rio Grande and the Colorado. From that time to the year 1872, cholera did not show itself on the continent of North America.

The disease reached *Central America* from New Orleans in the end of 1866; it spread from *Nicaragua* to Honduras, where it continued to the year 1868.

It was in the course of this epidemic that cholera appeared for the first time in the *States of the River Plate* and on the *West Coast of South America*. It was imported in some manner not yet explained, but it appeared first in April, 1866, in the army of Paraguay. The troops had for more than a year been watching the combined forces of Brazil and the Argentine Republic—who had taken the field against Paraguay—opposite the position which the latter had taken up on a small strip of marshy land at the confluence of the Paraguay and the Parana. The disease soon communicated itself from the Paraguayan army to

the soldiers of the confederate forces whom they came in contact with in engagements, and it spread rapidly through both armies. In autumn the pestilence was carried by vessels from the camp to Corrientes and places in the neighbourhood of that town; but its extension to Monte Video and Buenos Ayres was checked by the strict quarantine rules which those seaports enforced, and perhaps also by the intervention of the winter season. In January of next year (1866-67) the disease appeared anew among the troops, and also in Corrientes, whence it spread to La Paz, Bella Vista, Parana, and Rosario da Santa Fé, reaching Buenos Ayres in December. On this occasion they again succeeded, by a strict cordon, in keeping the disease out of Monte Video and all towns in Uruguay, and the winter following (1867) brought the epidemic a second time to an end. In the summer of 1867-68 the disease broke out afresh over the whole of the territory that had been already affected by it; and this time it succeeded in reaching Monte Video, and spread along the Uruguay to all the towns and stations that were in communication with the capital. On the setting in of winter (1868) and the suspension of hostilities, the cholera again came to an end. But towards the close of the year (1868-69) it broke out in certain inland provinces of the Argentine Republic, and, extending thence along the routes of trade and travel, it came to *Bolivia* and *Peru*, in which countries it spread down to the coast. In connexion with this epidemic in the states of the Rio de la Plata, stands the outbreak of cholera in *Brazil* in 1867. In April of that year the disease was imported from Paruguay to Santa Catarina, whence it spread through the provinces of Rio de Janeiro and Rio Grande do Sul, especially to the seaports of Porto Alegre, San Pedro de Rio Grande, Pelotes, &c., and along the banks of the river to São Jeronymo, Triumpho, Taquary, and even farther. In 1868 the cholera was again prevalent in Brazil to a not inconsiderable extent; but since then not only that country, but the whole continent of South America has been entirely free from it.

While cholera was thus gradually spreading from Arabia in a westerly direction as far as the shores of the Pacific, it had been extending at the same time northwards over the whole of Nearer Asia, and to the south-west over a great part of Northern Africa. In *Arabia* itself the pestilence had spread, doubtless in connexion with the homeward trains of pilgrims, into the central regions of Nedjed, and thence to the East Coast and towards the Shatt-el-Arab; and in the following year also it had been prevalent at numerous points in the Hedjah. As early as August, 1865, it had reached the shores of the Persian Gulf, and it then entered *Persia*. It appeared first in Bushire, Shiraz, and other places in the southern part of the province of Farsistan; then, in June of the following year—having been imported from Kurdistan—it was in the provinces of Aserbeidshan, Gilan, Mazanderan, and Chorassan, passing southwards through Teheran to Kashan. These outbreaks of cholera kept recurring during the six years following, at the most diverse points in Persia, so that it was not until the end of the year

1872 that the country was completely freed from it. Whether in this continuance of the disease in Persia during eight years we should recognise a connected series of epidemic outbreaks, or whether we should attribute the later outbreaks (particularly those of 1871 and 1872) to a new importation from Arabia, I am unable to decide from the data before me.

Proceeding in another direction, the cholera spread upwards from the Shatt-el-Arab in the summer of 1865 along the banks of the Euphrates and Tigris through *Mesopotamia* to Bagdad; while, by a second route, it penetrated from Syria towards the north of the country, reaching Urfa and Diarbekir, whence it proceeded down stream along the Euphrates and Tigris, and so gradually overran the whole country. In the next two years cholera was again epidemic at numerous points in Mesopotamia, while in 1868 and 1869 it was found only at isolated places and of a mild type. It was not until the autumn of 1870 that it again attained a wide distribution in the basin of the Tigris and Euphrates, having been imported probably from Persia. We come, lastly, to *Syria*, which was infected from Alexandria in 1865. The disease broke out on the 29th of June at Beyrout, and on the 1st of July at Jaffa, at each place a few days after the arrival of a ship from Alexandria with cholera on board; it spread quickly to Gaza, Ramleh, Jerusalem, Damascus, and Aleppo (where the first case occurred on the 11th of August), and penetrated thence, as we have seen, into Mesopotamia. Next year cholera was still epidemic at various points of Syria, at Tiberias, Jerusalem, Aleppo, &c., but during the four subsequent years it was absent from that country altogether.

On the continent of *Africa* cholera broke out, as already stated, at the beginning of 1865, on the coast of *Somali Land*, having been most probably imported direct from Bombay; from the Somali coast it spread into the interior. On that occasion, *Zanzibar* escaped; but cholera appeared there in October, 1869, and, according to trustworthy information, it was imported from the territory of the Wamassi, a nomadic pastoral people who appear to have got it from Central Abyssinia by way of the Gallas country. The pestilence wrought frightful havoc at first in the town of Zanzibar, and afterwards spread (in December) southwards to Cape Delgado, northwards to Mombas, Melinda, and Lamu, from Mombas into the Wanika region and to Brava, further to Madagascar, Nossi-Bé, and the Comoro Islands, and by caravan routes into the interior. At a later date (1870) the Mozambique coast also was attacked; scattered cases occurred on it during the whole of the following year, and in January, 1871, a new epidemic sprang up there, with which the outbreak the same year in the Seychelles, and on Nossi-Bé was perhaps connected. The *Mauritius*, in which cholera had been epidemic in the spring and summer of 1867, was not visited by it on this occasion.

Egypt, as we have seen, was the first point infected from Mecca; cholera broke out there some twelve days after the arrival of the first train of pilgrims, at Alexandria on the 2nd of June, and at Cairo on

the 17th of June. It spread through Lower and Middle Egypt, appeared at the end of the month in the province of Minieh, travelled up the valley of the Nile into Upper Egypt, and penetrated finally to Nubia, from which there are accounts of its prevalence during the summer of 1865 in Suakim, Cassala, and Taka. In the summer and autumn of the next year, cholera was again epidemic at several points in Egypt, at Cairo, Tantah, Port Said, &c. The outbreak on the coast of *Abyssinia* in October, 1865, is probably connected with the epidemic in Nubia; according to information derived from the English missionaries who were kept in captivity by King Theodore, the disease committed great ravages in the following year (1866) in the district of Tigré and still farther into the interior of Abyssinia; and it is this epidemic that appears to have been the point of departure, as we have seen, for the extension of cholera to the country of the Gallas.

Whether the disease travelled from Egypt in a westerly direction, or how far it may have done so, cannot be decided from the data before us. It reached *Tunis* first in the beginning of 1867, and it is said to have been imported by a band of smugglers from Sicily. The disease appeared first in the neighbourhood of the capital, and afterwards in Susa; it then spread over the whole plain, being aided by the movement of troops, and extended through the mountainous country and across the Algerine frontier to Biskra. *Algiers* had been already infected from France in the autumn of 1865. There is no definite information as to the extent of the epidemic there in that and the two following years; but it appears to have been especially prevalent in 1867, in which year the loss of life from cholera was estimated at 80,000. It died out at the end of the year, and since that time it has not been observed either in Algiers or in Tunis. The cholera had been imported into *Morocco* at the beginning of the pandemic in 1865, by pilgrims returning from Mecca, but, in that year, it does not appear to have spead far. However, in the summer of 1868, it broke out with great virulence, probably in connexion with the Algerine epidemic of 1867, spreading from the interior to the coast and attacking almost every locality in turn. The last offshoot of this epidemic on African soil is represented by the outbreak of the pestilence in November, 1868, in *Senegambia*, a country that had hitherto escaped the cholera. The first appearance of the disease was on the arrival of a caravan from Morocco at Podhor, an inland trading place on the Senegal, and the emporium of the overland trade between Senegambia and Morocco. Thence it spread both up and down the stream, and so reached St. Louis; from the latter point it was carried by natives from the Jolof country (between the Senegal and the Gambia) into the upper basin of the Gambia, where it likewise spread from place to place and so came ultimately, in March, 1869, to McCarthy's Island, and in May down the river to Bathurst. From Bathurst the disease was conveyed along the coast, northwards to the peninsula of Dakkar, and in a southerly direction as far as the mouth of the Rio Grande (Cacheo, Bissao and other places), a limit which was reached at the end of 1869, and beyond which cholera has not yet advanced.

Remission in 1869 *and* 1870.—The years 1869 and 1870 form a resting period in this pandemic for those countries that had suffered severely from the disease. During those years, cholera persisted at only a few points of the globe, out of India. But it is not to be regarded as having been altogether extinguished; and there is certainly no need to assume a new importation of the poison from India in order to account for the fresh general outbreak of the disease in 1871. Rather is it a repetition of essentially the same drama that was enacted in the third pandemic of cholera, when the disease subsided in 1851 and 1852, to revive in 1853.

On European soil, *Russia* remained a centre of cholera infection throughout. Although only in isolated cases or in small epidemic outbreaks, the disease still existed at various parts of the country (at St. Petersburg, at Moscow, in the Government of Kiev, particularly in the district of Lipowez, and elsewhere). In the summer of 1869, it reappeared at Kiev, and on that occasion it attained a more considerable compass than the year before, showing itself in the last months of the year in the Governments of Minsk, Toula, Orel, Poltava, Koursk, Smolensk, Moscow, Kalouga, and Novgorod, while even at St. Petersburg there were thirty-five cases of cholera officially reported. At most of the affected places, the disease survived the winter, and increased in the summer of the following year (1870) to an epidemic which spread over thirty-seven Governments, showing most severity in Orel, on the Don Steppe, and in the Governments of Ekaterinoslav and Astrakhan. On this occasion, again, the disease did not die out completely during the winter. It showed itself in epidemic form at various parts of the country in the first months of the following year (1871), and in summer it developed into one of the severest epidemics that Russia has ever suffered from. The area of the disease stretched from the shores of the Black Sea to Archangel, and from the western regions of Siberia (Governments of Tobolsk and Tomsk) to the western frontier of Russia in Europe. The number of fatal cases was reckoned at nearly 130,000. The Governments that suffered most were Tambov (with a mortality of 6·7 per 1000 inhabitants), Jaroslav (6·2 per 1000), Moscow (5·6 per 1000), Mohilev, Voronej, and Wilna (each with 5·0 per 1000); next to these came Orenburg (4·8), Smolensk, Vladimir, and Saratov (with 3·7), while Samara, Riazan, Penza, St. Petersburg, and Toula had a smaller death-rate. It will thus appear that the districts situated in the heart of the country suffered most. The disease again survived the winter in some of the affected districts, especially those on the western side, and in the spring of the following year (1872) an epidemic again developed which was almost as severe as that of the year before, the mortality being estimated at 120,000. This time it was especially the southern and western parts of the country that were visited—the

Governments of Astrakhan, Ekaterinoslav, Kherson, Bessarabia, Poltava, Kiev, Podolia, Volhynia, Tchernigov, Grodno, Minsk, and Poland. Except in the last, the disease died out over the whole country on the setting in of winter. In Poland only, it persisted through the winter, rising in the spring of the next year to a disastrous epidemic (the number of fatal cases being estimated at 30,000) which continued, with a winter remission, to the end of 1874. In Russia itself there were only scattered epidemic outbreaks in 1873 (particularly in the Governments of Grodno, Minsk, Volhynia, and St. Petersburg), the total number of deaths from cholera that year in Russia being only 4500; while in 1874, the country was quite free from the disease and has remained so ever since.

The diffusion of cholera on European soil during the years 1871-73 is connected directly or indirectly with the outbreak of the disease in Russia. From Poland the disease entered *Austria* in 1872. It appeared first in Galicia (as early as December, 1871), and in the spring of 1872 it spread thence to Moravia, Bohemia, and Austrian Silesia, and southwards to Hungary. In all those countries the epidemic lasted through the winter, and in 1873 it prevailed with greater intensity and over a wider area, spreading in particular from Hungary to Slavonia and Dalmatia, and to Lower Austria, where, however, it occurred in epidemic form in Vienna only. The cholera epidemic in Hungary in the years 1872 and 1873 is believed to have carried off the enormous number of 190,000 victims. At the end of 1873 the cholera disappeared throughout the whole of Austro-Hungarian territory, and has not been prevalent there since that time. The disease reached *Germany* in the summer of 1871, by importation from Russia to Prussia; in July it broke out in the governmental departments of Gumbinnen and Königsberg, in the beginning of August at Danzig, Stettin, Berlin, and Hamburg, shortly after in the departments of Posen, Potsdam, and Marienwerder, and in September at a few places in the bailiewicks of Lüneburg and Stade; but, except in East Prussia, it attained on the whole only a limited distribution, and in November it died out. In the following summer cholera re-appeared at Insterburg, Berlin, Gumbinnen, Thorn, Danzig, and other places in the eastern districts of the country, but only in isolated cases. In 1873, however, it was very destructive in Germany, having been imported from Galicia, Russia, and Austria (Vienna), and it spread so widely as to recall the worst cholera epidemics that the country had ever suffered from. Apart from an outbreak of the disease of unexplained origin at Magdeberg in April, and an epidemic probably connected therewith in Dresden and neighbourhood, the pestilence took its rise over the greater part of the most severely affected districts between the first weeks of June and the middle of July. It broke out almost at the same moment in all the eastern departments of Prussia (Posen, Bromberg, Marienwerder, Danzig, Oppeln, Breslau), a little later in Hamburg, in the province of Upper Bavaria, in Berlin, in the departments of Königsberg and Gumbinnen, and in the end of July, in the departments of Potsdam and Stettin, gradually overrunning the whole

of Germany, so that only a few small districts of the country escaped altogether. Throughout the whole empire, as far as can be ascertained, 1591 localities suffered from the disease, and of these 433 had the disease in epidemic form. The total ascertained number of deaths from cholera amounted to 33,156, of which 28,790 belonged to Prussia, 2612 to Bavaria, and 1005 to Hamburg. The departments that suffered most were Bromberg (with a mortality of 8·03 per 1000 inhabitants) Marienwerder (with 6·43), Königsberg (with 5·54), Magdeburg (with 5·45), Danzig (with 3·74), Hamburg (with 2·96), and Upper Bavaria (with 2·37). Toward the end of the year the disease was extinguished almost everywhere; only in Bavaria and in Upper Silesia (department of Oppeln) did it survive the winter, and at the last-named point it increased again in the spring of 1874 to an epidemic which exceeded in severity that of the previous year and lasted to October. Therewith ended the pandemic in Germany and in Europe, and since that time the continent has been entirely exempt from the disease.

An importation of cholera in another direction took place in 1871 from Russia to the *Danubian Principalities* and to *Turkey*. The disease showed itself first (in August) at Constantinople (where it grew to an epidemic that continued until January, 1872), and at a few points in Anatolia; in October it was at several places on the Danube, and, in November, on the north coast of the Black Sea, especially at Trebizond. Except at Constantinople, its intensity was everywhere slight. The disease prevailed to a somewhat greater extent in the summer of 1872 (June to September) in Roumania, especially at Jassy, but it was in the following year (1873) that Roumania suffered most severely; and in that year also it spread from the Danube over Bulgaria, and thence to a few points in the southern provinces of Turkey as far as Salonica. So far as the scanty information about this epidemic in the Turkish provinces warrants an opinion, its extent appears to have been but small. This group of countries has not been visited by cholera since the end of 1873.

During the last re-kindling of cholera in 1871-73, the South and West of Europe, as well as the Scandinavian kingdoms, suffered little from it. *Denmark* escaped altogether. In *Sweden* scattered cases of cholera were observed in 1871 at various points, and in the summer of 1873 the disease showed itself as a feeble epidemic in Helsingborg and in the district of Högenäs. At the same time (1873) *Norway* also was visited by a slight epidemic, confined exclusively to Bergen. During the severe epidemic of 1873 in Germany, cases of cholera were several times introduced into *English* ports without leading to a further extension of the disease. In *Belgium* none but sporadic cases were observed in that year, and these especially at Antwerp. In the *Netherlands* also, the disease occurred at various places (Bergen op Zoom, Dort, Rotterdam, Utrecht), but only sporadically or in small groups of cases, and without assuming a truly epidemic character. But in *France* the cholera in 1873 attained a somewhat greater importance, especially in the department of Calvados, where Caen in particular suffered heavily; scattered cases

occurred also at Havre, Rouen, and other localities in the department of the Seine-Inférieure, as well as in the departments of Manche, Morbihan, and Seine-et-Oise, and in Paris, but the disease nowhere developed to a great epidemic.

Cholera came to the continent of *North America* in November, 1871, by a ship with German emigrants, among whom cases of the disease occurred on the voyage. The vessel put into Halifax, and soon after her arrival cases of cholera, mostly running a fatal course, occurred in a small fishing village near the roadstead. The attack was confined to two labourers who had boarded the vessel, and to certain relatives who had come into direct contact with one of the two, or with one of those who sickened after him. The disease spread no farther that year, neither at Halifax nor in North America generally. Not until 1873, after importation of the disease in February to New Orleans, did an epidemic of cholera develop in that city, which again spread, as in 1866, along the Mississippi and its tributaries, and traversed a great part of the interior plain of North America. It was most severe in Louisiana, Alabama, Arkansas, Tennessee, Kentucky, Missouri, the western parts of Virginia, Ohio, Illinois, and Indiana; next in order, but only to the extent of scattered cases, in Iowa, Utah, Dakota, and Minnesota, while the Atlantic Coast States were almost entirely exempt. On this occasion it was again the towns of Memphis, Nashville, and St. Louis that had the severest visitations.

Just as the extension of cholera in 1871-73, over the whole of the territory here spoken of, stands in direct or indirect relation with the fresh epidemic outbreak of the disease in Russia in 1869 and 1870, so did Persia in 1870 form the point of departure of a pestilential progress which travelled through Nearer Asia and Egypt in 1871 and 1872. With the exception of sporadic cases in 1869 and 1870 at a few places in Mesopotamia, there was no cholera in Nearer Asia and Egypt from the end of 1867 to the beginning of 1870. It was only in Persia that the disease was epidemic during the whole of that period, more or less widely from time to time. Towards the end of 1870 it began to commit ravages in the southern province of Kerman and at Teheran, it appeared in November at Bender-Abbas, in February of next year (1871) at Bushire, and it then spread, on the one hand, along the east coast of Arabia, and, on the other hand, along the Euphrates and Tigris through a great part of Mesopotamia as far up as Mossul. The epidemic in Mesopotamia died out in January, 1872; in Persia, where the disease had been widely prevalent in 1871, it appeared at a few points in the following year, but only to a slight extent, and since 1872 cholera has shown itself no more in these parts. The course of events was much the same also in Arabia and Turkestan. As we have already seen, the disease reached the east coast of *Arabia* at the beginning of 1871, having been imported from the south coast of Persia; to the west coast of Arabia it was brought by a caravan from Irak Arabi (Mesopotamia), which first infected El-Haïl, the capital of the district of Jebel Shomer (in the inland province of Nedjed), whence the disease travelled

along the highroad to Medina, and from there to Mecca and along the strip of coast of the Hejaz as far as Hali. During the winter the sickness appears to have ceased, but in February of the following year (1872), it broke out afresh among the pilgrims assembled at Mecca, and was conveyed partly by them on their return, and partly by the movement of troops, to Hodeida, Jambo, Medina, and other places. By the end of April the disease had died out at all those points, and since that time (to 1881) Arabia has remained, as we have said, free from cholera. The re-appearance of cholera in 1872 in *Turkestan* and *Bokhara* is in all probability to be referred to importation from Astrabad (Persia). Again, the outbreak of the disease in *Nubia* in 1872 is said to have originated from Arabia. According to an official account, the disease broke out in the Nubian port of Suakin after the arrival of Turkish troops from Hodeida, and it spread thence over a great part of the provinces of Berber, Taka, and Dongola; by the end of the year the epidemic appears to have been extinguished, and it has not shown itself in subsequent years (until 1883) either there or in any other part of Egypt.

Finally, we must regard as a quite isolated phenomenon the outbreak of cholera in *Syria* in 1875. The origin of that epidemic is veiled in complete mystery. The disease appeared first (in March) at Hamah (in the upper basin of the Orontes); it spread thence ranging widely over the whole of the interior and the coast, and penetrating even into the Lebanon; and it did not die out completely until the end of the year. Among the inland places attacked were Aleppo, Aintab (in the north), and Damascus (in the south), and, among coast towns, Antakia, Latakia, Tripolis, Beyrout, and Saida may be mentioned. Nothing is known of an extension of the disease across the frontier to Mesopotomia or Anatolia.

Cholera in India in recent years.—Apart from the isolated epidemics of cholera in Russia and Upper Silesia in 1874, and in Syria in 1875, the disease became extinct as a pandemic in 1873. From 1875 to the end of 1880 it had shown itself at no point of the globe out of India. In *India* itself it has become a far reaching epidemic several times within the last decade, particularly in 1869 and 1870, 1872-73, and most recently in 1875. The accompanying table, which gives the mortality for the years 1871-75 in the several presidencies and provinces of the country, will enable us to estimate the prevalence of the disease.

Deaths from cholera in the various provinces of British India 1871-75.

	1871	1872	1873	1874	1875
Punjaub . . .	369	8,727	148	78	6,246
N.W. Provinces .	3,473	50,565	15,268	6,396	41,106
Oude . . .	16,032	26,566	3,961	68	23,321
Bengal . . .	20,396	46,901	64,366	56,876	112,276
Central Provinces .	19	1,592	344	14	14,643
Berar . . .	581	1,578	—	2	22,465
Bombay Presidency	5,855	15,642	283	37	47,573
Madras Presidency	17,656	13,247	840	313	94,547
British Burmah .	162	640	8,109	960	761
Ceylon . . .	?	?	14	—	1,817

§ 103. Countries that have Escaped Cholera.

The sketch here given in main outlines of the history of cholera shows that, since the first general occurrence of the disease in 1817, it has with each successive pandemic obtained a wider geographical distribution, and that its area now covers the greater part of the inhabited globe. Among the larger territories or island-groups which still enjoy a complete immunity from cholera, are the continent of Australia,[1] the islands of the Pacific Ocean, the East Coast of Africa south of Delagoa Bay, the Cape of Good Hope, the southern and central divisions of the interior of Africa up to the Soudan, the West Coast of Africa as high as the Rio Grande, and the islands of St. Helena and Ascension. In South America the exempted territories are the South Polar lands, the Falkland Islands, Terra del Fuego, Patagonia, and the republic of Chili ;[2] in North America : the whole country north of the 50th parallel, and the Bermudas ; in Europe : Iceland, the Faröe Islands, the Hebrides, the Shetland and

[1] The statement that cholera prevailed in 1832 on the west coast of Australia ('Gaz. méd. de Paris,' 1832, p. 499) rests upon hardly reliable newspaper information.

[2] The assertion that Valparaiso has been visited by the cholera rests on an error, as now appears from the most recent communications of Bleihöfer (in the 'Nachrichten über die Gesundheitszustände in verschiedenen Hafenplätzen,' Heft xi, Hamburg, 1877, p. 34).

Orkney Isles, Lapland, and the Russian territory north of the 64th parallel; in Asia: the northern governments of Siberia, and Kamtschatka. Whether cholera has penetrated to Mongolia and Manchooria is a point that I have not ascertained.

Within those countries and islands that have been visited by cholera, there are, it is true, many regions of greater or less extent which have hitherto escaped the pestilence, or have been only slightly touched by it; as, for example, Switzerland, several mountainous districts in the south-east of France, the south-west of Germany, especially Baden and Würtemberg, the northern districts of Scotland, and large parts of Greece. This absolute or relative immunity of the regions named is in part owing to certain local conditions, to be afterwards referred to, which form an insuperable obstacle to the disease becoming an epidemic; and there are examples of the same, on a small scale, in numerous localities which have been repeatedly encircled by severe epidemics of cholera, and yet have remained free from the disease, notwithstanding that communication with the infected districts was not only not interrupted, but had even become more frequent owing to fugitives from those districts, whether the healthy or the sick, resorting to them. But that immunity is explained, to a certain extent also, by the circumstance that a large part of the exempted tracts of country are either entirely or to a great extent remote from the lines of traffic, no importation of the morbid poison therefore ensuing. Finally, there are many places hitherto exempt which owe their protection from cholera partly to good fortune; and this last is a factor which we shall be disposed to pay all the more heed to in judging of the question, inasmuch as experience has shown that many districts which were spared by the earlier epidemics have been visited by the later.

§ 104. Its Native Habitat.

There underlies this account of the distribution of cholera in space, the assumption that the disease did not arise *de novo* at every place where it has shown itself, but rather

that it is indigenous at certain points on the earth's surface, and that its extension beyond those points depends on the conveyance of the specific agent that gives rise to it, or, in other words, of a poisonous substance. There can be now no reasonable doubt as to the correctness of this view of the facts; and, further, it cannot be longer questioned that the *home of cholera* is to be sought for in India, and perhaps also in Lower India and the Indian Archipelago.

The view that cholera may, under certain circumstances, arise also autochthonously beyond those territories, is supported more particularly by two arguments. The first of these is that, at the time of the pandemic prevalence of cholera, there were many instances of its appearance as an epidemic in extra-Indian countries in which no infection of the plague-stricken district could be traced to a source outside. The second is the fact that, from the earliest times, and at all times, the disease has been repeatedly observed either in sporadic or epidemic form at the most various points of the globe, without the very slightest connexion with influences from India having occurred or having even been assumed. Hippocrates, Celsus, Aretaeus, Cælius Aurelianus, and other Greek and Roman physicians, it is contended, had seen and described cholera; all the Arabian and Arabistic writers on medicine mention the disease; and there have come down to us from the sixteenth to the eighteenth centuries, a not inconsiderable series of accounts of cholera epidemics[1] which leave no doubt as to the existence of the disease long before 1817, and furnish proof of its autochthonous development, inasmuch as they show that it had been isolated within narrowly circumscribed areas. But the weakness of each of these arguments will be brought out by an unprejudiced examination of the facts. The error underlying the first will appear when we treat of the mode in which cholera spreads. The second rests upon a mistake in diagnosis; upon the confounding of two forms of disease, which certainly approach one another closely in the matter of symptoms, but, as regards their origin and clinical history, have nothing in common. They differ,

[1] A very complete summary of all these observations will be found in Macpherson's 'Annals of Cholera,' pp. 15, 78.

indeed, so essentially that the one—the cholera Indica—is a distinctly communicable disease with a mortality, moderately estimated, at 50 per cent., while the other—the so-called cholera nostras—has not exhibited a single trace of communicability, and runs a course which is never fatal except under certain peculiarly unfavorable circumstances, such as extremes of age, debilitated constitution, and the like.

§ 105. Cholera in India and Elsewhere previous to 1817.

The accounts of the *endemic and epidemic prevalence of cholera* (Asiatic) *in India* reach back to the remotest times. But the oldest notices of the matter, which occur in the historical and medical writings of the Hindoos,[1] and in the reports of travellers, whether medical or non-medical, of the sixteenth and seventeenth centuries,[2] are to be used with caution in deciding this question, inasmuch as cholera is plainly mixed up in them with other forms of disease,—with colic and dysentery, and especially with cholera nostras. Those accounts have still their value, in so far that they show that a form of disease, bearing the name of *Mordeshi*, has at all times been prevalent in India, either sporadically or epidemically, being marked in its course by phenomena known to us as those of cholera, and in most cases, ending in the death of the patient often within a few hours.

The first reliable information about Asiatic cholera that I have found, occurs in the account given by Sonnerat[3] of a disease that was prevalent in 1768 and 1769 in the neighbourhood of Pondicherry and over the whole Coromandel coast, and which carried off 60,000 victims in one year.

[1] The often-quoted passage from the Ayur Veda of Suçruta, which is adduced as evidence of the occurrence of cholera in India in the remotest antiquity, and upon which, for example, Wise relies ('Commentary on the Hindoo System of Medicine,' Calcutta, 1845, p. 330), appears from the translation of Liétard (given by Scoutetten in 'Gaz. hebdom. de Méd.,' 1869, No. 29, p. 452) to furnish a picture of the disease that is, to say the least, an ambiguous one; and that objection is entirely apart from the doubt as to the very great age of this writing, at least in the form in which it now exists, and the reasonable suspicion that its author or editor had made use of Greek writings as a model.

[2] Macpherson, l. c., p. 79, *et seq.*, gives a very complete summary of all these accounts.

[3] 'Voyage aux Indes orientales,' Paris, 1782, i, 113.

Next come the trustworthy accounts of an epidemic of cholera in 1781 in the French army of occupation under General Anderne, of epidemics the same year in Calcutta,[1] and in Madras, where the disease, according to the report of Curtis,[2] was epidemic the following year as well (1782); further, in 1783, on the Madras coast;[3] and among the pilgrims collected at Hurdwar, twenty thousand of whom are said to have died in less than eight days;[4] then at Arcot in 1787, Bellary in 1788, Travancore in 1792, and at various points in the Presidency of Bengal during the years 1811-13.[5] At the same time, numerous other medical chroniclers of that period from various parts of India draw attention, in their descriptions of the disease, to its endemic character;[6] but we find nowhere any reference to the communicability of cholera, an aspect of the disease which appears to have entirely escaped the observers prior to 1817, or prior to the outbreak of the first pandemic. It is precisely that circumstance which makes it difficult to answer the question whether the endemic seats of cholera should be looked for exclusively in India proper, or whether the disease may not be indigenous in other parts of Asia as well.

It can hardly be doubted that cholera had been epidemic in extra-Indian regions of Asia repeatedly before 1817. The sketch which Bontius[7] gives of the disease observed by him in *Java* in 1629, bears the perfect impress of Indian cholera; and there are accounts of the epidemic prevalence of the same disease there in 1689. In like manner, the accounts of cholera epidemics in *Ceylon* given by Curtis for the year 1782, and by Johnson[8] for the years 1790 and 1804, relate beyond doubt to Indian cholera. The reference of Zacutus Lusitanus[9] to the alleged occurrence of cholera in *Arabia* in the seventeenth century, is a doubtful one; and so also is the statement of Burke, in his report on the epidemic of cholera in the *Mauritius* in 1819, that a disease entirely similar had been prevalent there in 1775.[10]

It is impossible to decide whether cholera had been observed in Further India previous to the year 1819. The

[1] 'Bengal Med. Reports,' quoted by Macpherson.

[2] 'Account of the Diseases of India,' Edinburgh, 1807, p. 44.

[3] 'Madras Medical Reports,' in Macpherson.

[4] 'Bengal Medical Reports.'

[5] Macnamara, 'History of Asiatic Cholera,' London, 1876, p. 42.

[6] See Macpherson, l. c., p. 124, *et seq.*

[7] 'De medicina Indorum,' libr. iv, p. 69. Lugd. Bat., 1718.

[8] 'The Influence of Tropical Climates,' &c., London, 1815, p. 396.

[9] 'Praxis Historiarum,' lib. ult., Nr. vii, obs. 3. Op. omn. Lugd., 1667, ii, 631.

[10] Quoted by Tholozan in 'Gaz. méd. de Paris,' 1868, No. 41, p. 581.

statement that the disease is endemic in the territories bordering the Straits of Malacca is quite erroneous,[1] and the assertion of the French physicians[2] that cholera is endemic in Cochin China appears to be without foundation. But even for Ceylon, Java, and other parts of the Indian Archipelago, there is not the smallest proof that indigenous foci of cholera occur at any point whatever in those regions; all the epidemics of cholera that have happened in Ceylon and the East Indies coincide with widely-spread outbreaks of the disease on Indian soil, and an early importation into those regions, in due course, is readily intelligible from the close intercourse in which they have always stood with the Indian continent. It appears from the reports of Dutch physicians that many islands of the Indian Archipelago enjoy a very decided immunity from cholera. Thus, Timor has remained hitherto undisturbed by the disease;[3] in Macassar it has rarely been seen;[4] in Borneo it has occurred only four times in all up to 1872;[5] in Banka it appeared for the first time in 1852;[6] Amboina likewise has escaped almost entirely hitherto;[7] in Manilla cholera had not been seen of recent years,[8] [until the severe epidemic of 1882]; and the Nicobars have shared this immunity from the disease since 1831.

The opinion that *China* had been visited by cholera (Asiatic) in pre-Christian times is based upon accounts of

[1] Macnamara ('History,' p. 141) says: "I have carefully examined the returns of the health of our convict, civil, and military populations, which in former times were sent regularly from the Straits' settlements to the Medical Board in Calcutta, and I find with regard to Singapore, Penang, and Malacca, from 1827-40, that not a single death from cholera occurred either among the troops or convicts at any one of these stations throughout this period." In an official report on the diseases of Singapore ('Madras Quart. Med. Journ.,' 1839, i, p. 71) we read:—"As an epidemic cholera is unknown at Singapore;" and in a communication by Dick ('Army Med. Report,' 1873, vol. xv, p. 330) it is stated that cholera has never been prevalent in Pulo-Penang (Prince of Wales Island).

[2] Chabassu, in the 'Union Méd.,' 1863, No. 90; Laure, 'Hist. de la Marine Franç.,' &c., Paris, 1864, p. 99; Richaud, in the 'Arch. de méd. nav.,' May, 1864, p. 340; Gimelle, 'Union Med.,' 1869, No. 53 (Feuilleton).

[3] 'Arch. de méd. nav.,' 15th July, 1870.

[4] Ib., April, 1871, p. 247.

[5] Ib., Jan., 1872, p. 21.

[6] Ib., Feb., 1873, p. 102.

[7] V. Hattem, in the 'Nederl. Tijdschr. voor Geneesk.,' 1858, p. 538.

[8] Taulier, in the 'Arch. de méd. nav.,' Dec., 1877, p. 401.

the disease in a Chinese medical writing whose date is referred by Livingstone, to whom we are indebted for a detailed report upon it,[1] as far back as the Hippocratic period, and which first appeared in type in 1790. The description of the symptoms of the disease, according to Livingstone's English translation, is as follows:

"The Ho-lwan (cholera) is a sudden attack of pain in the heart and abdomen, with vomiting and purging, a dread of cold and a desire of warmth. It is accompanied with pain in the head and giddiness. When the pain attacks the heart first, vomiting comes on first; when the pain commences in the abdomen, the purging precedes. When the pain in the heart and abdomen synchronise, the vomiting and purging come on at the same time. When the disease is severe, the patient has spasms; and when these enter the abdomen death ensues."

To my thinking, it requires an abounding faith to find the picture of cholera in these words; and Livingstone himself remarks that an old Chinese doctor, who was witness to the general outbreak of cholera in Bengal in 1817, stated, in answer to questions, that he had treated many cases of this same "Ho-lwan" in China in the course of the previous thirty years, but had lost only about three patients in the hundred. Milne,[2] who lived many years in China, and had made very thorough inquiries into the occurrence of cholera (Asiatic) in that country, arrived at the conviction that the disease had never been epidemic in China previous to the general outbreak of it in 1820. We shall find less occasion to seek for endemic foci of cholera in that country if we bear in mind that China (with Japan) has entirely escaped the disease from 1863 [down to the epidemic of 1881-82].

The question of the home of cholera, therefore, centres really in the inquiry concerning its endemic prevalence in India. From no quarter has any question been raised whether the native habitat of the disease may not extend over the whole peninsula; it is admitted that cholera, as an endemic, is limited to certain portions of the country, and perhaps even to a few points only. But to determine those

[1] 'Transact. of the Med. Soc. of Calcutta,' 1825, i, 204. This is probably the source from which Dudgeon also has drawn, when he states that cholera is a primeval disease of China ('Glasgow Med. Journ.,' 1877, p. 322).

[2] 'Edinb. Med. and Surg. Journ.,' April, 1844, p. 483, and 'Life in China,' London, 1857, p. 517.

regions or points with certainty, we may take to be beyond the bounds of possibility, inasmuch as there is no sure criterion to go by. It stands to reason that its more or less frequent epidemic prevalence is not decisive by itself. Its annual occurrence either as an epidemic or in sporadic cases would be more significant. But it is just in relation to this matter that the data before us from the various parts of India are wanting in the completeness and trustworthiness that would justify a decided conclusion.[1] Thus, in laying down the boundaries of the endemic foci of cholera, we shall not arrive at more than a probable estimate, or indicate them otherwise than in broad outlines. In Hindostan, according to the researches of Bryden,[2] it is the region bounded on the east by the 91st or 92nd parallel of longitude, on the west by the 81st parallel, on the north by the latitude of 27°, and on the south by the shores of the Bay of Bengal (including the delta of the Ganges and the territory at the mouths of the Mahanuddy) that forms the proper native habitat of cholera. This gives a focus of disease which extends from the mountainous districts of the Brahmaputra to the hill regions of Rajmahal and Cuttack, and, on its northern border, along the Terai from Lower Assam to the district of Purnea. According to Macnamara's inquiries[3] its western boundary extends still farther, being formed by a line running from Saugor (Central Provinces), through Allahabad and Gorackpore, to the slopes of the Himalaya. Within this region, it is the eastern districts, especially those of Calcutta and Dacca, that experience the worst visitations; towards the west, the intensity becomes less and less; and

[1] How fallacious may be the conclusions obtained by that mode of investigation is shown by the fact that Lower Bengal, whose importance as a centre of endemic cholera cannot now be doubted, possesses scarcely any records of cholera epidemics in previous centuries. "It so happens," says Macpherson ('The Early Seats of Cholera in India,' Lond., 1869, p. 30), "that historically Bengal is the part of India in which no accounts of the very early prevalence of the disease have been discovered;" and, again ('Annals of Cholera,' London, 1872, p. 183): "We know the medical history of Lower Bengal for that period tolerably well, and that during the last half of the eighteenth century there was little cholera in Bengal."

[2] 'Report on the Cholera of 1866-68 in the Bengal Presidency,' Calcutta, 1869.

[3] 'Treatise on Asiatic Cholera,' London, 1870.

in the Punjaub, the Rajpootana States, and Sind, endemic cholera does not occur at all. There appears to be no ground for the statement, often made, that the Madras Presidency is entirely free from endemic foci of cholera.[1] Macnamara and Montgomery[2] specially name several districts on the Coromandel coast (Nellore, Trichinopoli, Tanjore, and Madura) as indigenous seats of the disease, and Inglis[3] states that cholera is endemic at many points in the Presidency, as, for instance, Madras itself. Information is entirely wanting as to the endemic occurrence of cholera in the interior of the Deccan; and, for the Bombay Presidency, the only communications that I have met with on the subject are those of Crespigny[4] and Gorringe[5] from the districts of Rutnagherry and Ahmedabad, in Gujerat. However incomplete, therefore, our information may be as to the proper habitat of cholera in India, all that we do know points to Lower Bengal as the headquarters of the disease.

§ 106. Conditions Favorable to its Origin and Diffusion.

A glance at the diffusion of cholera in time and place, as here sketched in its main outlines, brings into prominence two points of view, from which must proceed our inquiry into the etiology of the disease, or, in other words, our discussion of the question, *What are the factors which determine the origin and promote the diffusion of cholera?* The one salient fact is, that the disease is indigenous at certain points in India which we know with tolerable accuracy, and that its occurrence beyond these points has always been associated with a conveyance of the cholera poison. The other fact is that the diffusion of the disease in that manner is not so extensive as the importation of the poison itself; that

[1] In the passage already quoted ('Annals of Cholera,' p. 183) Macpherson calls attention to the fact that, while Bengal was little affected by cholera in the period spoken of, there were severe epidemics on the Malabar coast; and there is little likelihood, as he adds, that the disease had been imported thither from other parts of India.

[2] 'Madras Quart. Journ. of Med.,' 1869, i, p. 396.

[3] 'Army Med. Reports,' 1863, p. 374.

[4] 'Bombay Med. Transact.,' 1859, n. s., iv, p. 94.

[5] Ib., 1861, n. s., vii, p. 107.

many larger or smaller regions of the globe, into which the virus has been repeatedly carried by commercial traffic, have not been visited at all by epidemics of the disease, or, at least, have not suffered on each occasion of its importation, but have, on the other hand, enjoyed either an absolute or a temporary immunity. From this it may be concluded that, as there are certain local peculiarities which furnish the conditions for the endemic disease, so also there are certain factors, residing in the circumstances of place or season, which are necessary to give potency to the cholera poison beyond its native habitat. The first, so far as I know, to do justice to this fact in all its comprehensiveness, and to give definite expression to it, was Hergt, who said:[1] "The rise of the cholera epidemic at any one place implies, besides importation of the contagium, certain local conditions of atmosphere and soil as well. Those conditions must be able, at a given place, to generate themselves and to disappear again."

After smallpox and influenza, we know no acute infective disease which has, on the whole, attained so wide a diffusion over the globe as cholera, from the tropics to extreme polar latitudes; and has carried with it, amidst all the differences of climate, soil, and social well being, so complete a stamp of uniformity in its fundamental characters, in its natural history, in its type, and in its fatality, and that, too, both in epidemics great and small, and in sporadic cases. Herein lies the proof that the conditions upon which depends the potency of the morbid poison outside its habitat, are met with in the widest diffusion over the earth's surface. And although the very narrow limits of its native territory show that there must be quite peculiar conditions existing there, in order to constitute the special factors in the morbid poison, conditions which do not reside *exclusively* in the atmosphere or the soil or the social life characteristic of the country; yet experience points to the same influences that have determined the epidemic occurrence of cholera beyond India, as having been of essential importance for the diffusion of the disease in that country also.

[1] 'Geschichte der beiden Cholera-Epidemieen des südlichen Frankreichs, &c.,' Coblenz, 1838, p. 162.

§ 107. Influence of Altitude.

Among the external factors which have helped more or less to determine the epidemic outbreaks of cholera, and on which the potency and reproduction of the cholera poison appear to depend, the first place must be assigned beyond doubt to certain *circumstances of locality*. In evidence thereof, is the frequently observed fact that, in every outbreak of the disease within a considerable territory, not only certain localities have been visited with particular frequency and with particular intensity, but that even particular quarters, streets, groups of houses, and indeed single houses, have formed a chief focus of the epidemic. Another piece of evidence, of the first importance, is the fact that, in many districts of greater or less extent, the cholera has never reached any considerable or strictly epidemic development notwithstanding repeated importations of the poison; those districts enjoy, accordingly, an absolute or relative exemption from this disease, such as can only depend upon local conditions.

Exempted places.—We find examples of this at the following places: Würzburg, according to the experience of 1866[1] and of 1873;[2] Frankfort-on-the-Main; Olmütz, where in 1866, as on all previous occasions, only an insignificant distribution of cholera occurred, notwithstanding the state of war then existing;[3] Falun, which has always remained free from cholera hitherto in spite of an active commercial intercourse with a highly infected neighbourhood;[4] Rouen, where the disease has never gone beyond a very small extent, although it stands in free communication with places that have suffered so much as Caen, Amiens, &c.;[5] Versailles, which is in constant communication with Paris by two railways, and has been the refuge of many thousands of fugitives whenever cholera has broken out in the capital, but has never yet suffered from an epidemic of it;[6] Lyons, which has often had the disease introduced

[1] Cf. Grashey, in the 'Würzb. Med. Zeitschr.,' 1867, vii, 135.

[2] Goek, in the 'Verhandl. der Würzb. phys.-med. Gesellschaft,' 1874, vi, 49. There were in all 120 more or less severe cases of cholera observed there in 1873, of which one half occurred in the Julius Hospital.

[3] Pissling, in the 'Wiener Med. Wochenschrift,' 1866, No. 86.

[4] Hallin, in the 'Nord. Med. Ark.,' 1870, ii, 53.

[5] Leudet, in the 'Bull. de l'Acad. de Méd.,' 1866, xxxii, 73, and 'Union Méd.,' 1873, ii.

[6] Guerin, 'Le Choléra à Versailles,' Paris, 1866.

in like manner by fugitives from neighbouring places, but has only once, in 1854, become the seat of a slight epidemic;[1] Sedan, exempt hitherto, although cholera has been prevalent all over the surrounding country;[2] Cheltenham, which has enjoyed a complete immunity from cholera under the same circumstances;[3] and Martinique, where merely sporadic cases have occurred, notwithstanding repeated importations.[4]

At the very first general diffusion of cholera in India in 1817-19, medical observers had their attention directed to the fact that the hill forts remained exempt in a remarkable way, while the disease was prevalent in the plains round about, and that the removal of troops from an infected district to an elevated station, without any separation of the sick from the healthy, was followed by a speedy disappearance of the disease from among them. Observations to that effect were made by Jameson,[5] Scott,[6] Orton,[7] Anderson,[8] Whyte,[9] and others, and they have often been confirmed in later times.

Thus, Spence[10] records that, among a body of troops which had been seized with cholera on the march from Bombay to Poonah, the disease died out as soon as they were taken to Kandallah, situated on the slope of the Ghâts, at a height of 3000 feet. According to a report of Lorimer's,[11] a detachment which had contracted cholera on the march from Secunderabad to Palaveram, and had suffered from it for four weeks, got rid of it as soon as they reached the Red Hills. Mouat[12] observed the same thing in the case of a detachment of dragoons on the march from Bangalore to Madras. Scoutetten,[13] also, when cholera was prevalent in Algiers in 1835, achieved a successful result by moving the

[1] Pettenkofer, in the 'Zeitschrift für Biologie,' 1868, iv, 400.

[2] Brégi, 'Essai sur la Topogr. méd. de la Ville de Sedan,' Paris, 1874, p. 30.

[3] Wilson, in the 'Brit. Med. Journ.,' 7th Sept., 1872, p. 262.

[4] Rufz, in the 'Arch. de méd. nav.,' June, 1869, p. 439.

[5] 'Report on the Epidemic Cholera . . . in the Presidency of Bengal in the Years 1817-19,' Calcutta, 1820, pp. 32, 72.

[6] 'Report on the Epidemic Cholera . . . in the Territories subject to the Presidency of Fort St. George,' Madras, 1824.

[7] 'Essay on the Epidemic Cholera of India,' Madras, 1820, p. 401.

[8] 'Edin. Med. and Surg. Journ.,' xv, July, 1819, p. 354.

[9] In the (Bombay) 'Reports of the Epidemic Cholera, &c.,' Bombay, 1819, p. 13.

[10] 'Lond. Med. and Surg. Journ.,' 1832, n. s., i, 61.

[11] 'Madras Quart. Med. Journ.,' 1839, i, p. 27.

[12] Ib., 1840, ii, 443.

[13] 'Gaz. hebdomad. de Méd.,' 1869, No. 49, p. 774.

troops to an elevated situation. Cazalas[1] did the same with the French troops quartered in the Dobrudscha in 1854; they were attacked by the epidemic to a disastrous extent, but the disease at once began to decline among them after they were taken to the hills. Whenever the cholera has broken out at Fort St. George (Madras), the same practice has repeatedly been put in force with success.[2] Referring to the statements of Cunningham on the cholera epidemic of 1872 in the North-West Provinces, Murray remarks:[3] "During this severe epidemic, the removal of troops into camp has, in some instances, been promptly and efficiently conducted, with the most satisfactory results. . . . . In some instances, the disease clung to the party, but in general it was shaken off."

The epidemic occurrence of cholera at very considerable *elevations* furnishes proof, however, that the height of a place or of a district does not of itself secure any immunity from the disease.

When the disease spread widely over India for the first time, it crossed the mountain range which separates Nepaul from Tirhoot, and reached an elevation of 4000 to 5000 feet in the lofty hill districts of Chatmandu and Putan, and of 3000 feet on the plateau of Malwa. In 1838 it reached as high as 8000 feet;[4] in 1845 to an elevation of 6000 feet at Kussouli near Simla,[5] and in 1856 to a height of 5000 feet in the Nepaul valley.[6] The plateau of Erzeroum, 6000 to 7000 feet in height, has been repeatedly visited by cholera. In 1850, the disease travelled in Grenada, along the banks of the Amazon, to Neiva and Bogota, or to an altitude of 7800 feet.[7] On the lofty table-land of Mexico, cholera has been epidemic on several occasions, as, for example, in 1850 at Puebla and in the City of Mexico; and, in several of those epidemics, it broke out in a most destructive form.[8]

But cases of that kind belong to the rare exceptions. Usually under such circumstances, the disease has found only a slight diffusion; the amount of sickness has almost always diminished in proportion to the height to which cholera has ascended above the plains, and it has been quite

[1] 'Examen théor et prat. de la Question relative à la Contagion du Cholera,' Paris, 1866, p. 28.

[2] 'Madras Month. Journ. of Med. Sc.,' 1870, ii, 459.

[3] 'Brit. Med. Journ.,' Jan., 1874, p. 73.

[4] Graves, in the 'Dubl. Journ. of Med. Sc.,' Jan., 1840, xvi, p. 366.

[5] Ireland, in the 'Edin. Med. Journ.,' Jan., 1863, viii, part 2, p. 613.

[6] Brown, in the 'Indian Annals of Med. Sc.,' July, 1858, p. 496.

[7] Wis, in the 'Gaz. méd. de Paris,' 1850, p. 554.

[8] Jourdanet, 'Le Mexique et l'Amerique Tropicale: climat, hygiène, et maladies,' Paris, 1864, p. 405.

common for the elevated points to remain almost or altogether exempt, in spite of numerous imported cases. Even in those instances where cholera has taken a wider range, there have been differences observable in the spreading power of the disease, corresponding to the higher or lower elevation of the several localities.

An interesting example of the last-mentioned law of cholera diffusion is furnished by the epidemic of 1856 in Nepaul, at a height of 5000 feet where, according to the investigations of Brown (l. c.), the conditions of cholera-infection in the various towns and villages were exactly in accordance with the law that was worked out by Farr (*vide infra*) for London. "Immunity from cholera," Brown concludes from his inquiries, "does not depend upon mere elevation in the strict sense of that term, but it depends upon the elevation above the place where cholera is raging, whatever the height of that place may be." This relative immunity of elevated localities, in contrast to low-lying places in their immediate neighbourhood, is confirmed by the experiences made on Mount Abu (5000 feet high, in the Arawalli range of Rajpootana),[1] and on the Shivaroy mountains (3000 to 4000 feet) in the Salem district,[2] where there has never been a proper epidemic in spite of repeated importations of cholera from places situated below. On the breaking out of cholera in California in 1850, the disease spread along the flats of the Sacramento and San Joaquim, while the more elevated parts of the country, and especially the mining districts, which were in free communication with the plains, were almost entirely exempted.[3] Polak[4] draws attention to the fact that, while cholera was prevalent in Teheran, it was carried from the capital to elevations of 6000 feet, but it occurred only in scattered cases, and at still higher elevations it did not occur at all, notwithstanding that many persons resorted thither. (The Shah of Persia, on three occasions when cholera was epidemic, located his camp, containing 10,000 persons, in the valley of the Laar at an elevation of 7500 feet.)

Those differences in the diffusion of the disease come out not less markedly in a comparison of adjoining districts with slightly different elevations, and they are even more clearly shown in such towns or villages as are situated *on a slope* or *in a basin*, and whose several quarters, therefore, present different altitudes.

[1] Lownds, in the Bombay Med. Transact.,' 1857, n. s., iii, p. 175; Ogilvy, ib., 1861, vi, p. 206.

[2] Cornish, in the Madras Quart. Journ. of Med. Sc.,' Oct., 1861, p. 313.

[3] Praslow, 'Der Staat Californien in med.-geogr. Hinsicht.,' Göttingen, 1857, p. 37.

[4] Report of the Commission of the International Sanitary Conference at Constantinople, p. 131.

At the first outbreak of cholera in Russia in 1830 and 1831,[1] attention was drawn to the fact that the disease, having avoided the spurs of land running in from the frontier mountains, spread particularly towards the deepest parts of the plains, where it attacked the villages situated in valleys, in preference to those on high ground; and the same thing has been observed to happen in later epidemics.[2] Many observations from Hungary[3] and Austria[4] tend to prove that the diffusion of cholera in those countries was after the same manner. Thus, when it broke out in Styria in 1832, it was confined exclusively to the flats, particularly to the villages in the valley of the Mur,[5] within the Graz circle. In Illyria on that occasion, as well as later,[6] it was chiefly prevalent in the more level Carniola, and much more rarely (proportion of 0·4 to 3·5 per cent) in the mountainous Carinthia, where it was epidemic at a few places in the Villach circle, but occurred only sporadically in the circle of Klagenfurt. In Lower Austria, the mountainous regions were scarcely touched by the epidemic of 1831.[7] In Moravia and Austrian Silesia, the number of places affected in the plains stood to those of a greater elevation in a proportion of two to one;[8] and the conditions were similar in Galicia, where, in the northern and low-lying circle of Lemberg, no place escaped the cholera, while in the elevated southern parts there were forty-five villages in which not a single case occurred.[9] In Germany this relation is very clearly expressed, not only in the relatively scanty occurrence of the disease on the south-western plateau as contrasted with its general diffusion over the low plains of the north-east, but also in the amount of sickness within small zones of either region, which are distinguished from one another by differences in their elevation.[10] Suerman's[11] words, with

[1] Lindgren, 'Der epid. Brechdurchfall beobachtet zu Nishninovgorod,' Dorpat, 1831, pp. 3, 29.

[2] Frettenbacher, 'Gaz. méd. de Paris,' 1849, No. 2, p. 23.

[3] Eckstein, 'Die epid. Cholera in Pesth im Jahre 1831,' Pesth, 1832; Flittner, in the 'Oest. med. Jahrb.,' 1832, u. s., iv, p. 222; Salawa, in the 'Ungar. Ztschr. für Natur und Heilkunde,' 1856, No. 38, p. 300.

[4] Report in the 'Oest. med. Jahrb.,' 1833, n. s., vi, p. 7.

[5] Report, ib., 1839, n. s., xx, p. 339.

[6] Melzer, 'Studien über die asiatische Brechruhr,' Erlangen, 1850, p. 21.

[7] Report in the 'Oest. med. Jahrb.,' 1833, n. s., vi, p. 7.

[8] Report, ib., 1839, n. s., xx, p. 536.

[9] Salwikowski, ib., 1832, n. s., iv, p. 552.

[10] On this subject see Burkart on the diffusion of cholera in Würtemberg ('Ztschr. f. Biol.,' 1876, xii, 366). In my report on the epidemic of cholera in Germany in 1873 ('Berichte der Cholera-Commission für das deutsche Reich.,' Berl., 1879, vi, p. 304) I have said: "On few points is there so complete an agreement among those who observed the recent epidemic as that the low situation of a locality, especially at the bottom of a basin-like formation, has been a true predisposing cause of an epidemic, while a relatively high situation has been a safeguard against it."

[11] 'Spec. med. de Choleræ asiaticæ itinere per Belgium septentrionale,' Traj. ad Rhen., 1835, p. 254.

reference to the diffusion of cholera in the Netherlands in 1832-34, were: "Altiores patriæ tractus fere a Cholera immunes mansere." In England it had been observed in the very first epidemic (1831-32), that the cholera was much rarer on the high-lying parts of the interior than on the coast, the banks of rivers, and other low-lying districts. It had, indeed, spared the highland country altogether, and when the same circumstance occurred in subsequent epidemics, an expression was found for it in arithmetical terms. In the epidemic of 1848-49, cholera was especially prevalent at nine centres in England, representing 136 districts; the population of these made up about four-tenths of the whole population of the country, and among these 46,592 persons, or 5 per 1000 inhabitants, died of the disease; in the remaining 487 districts, mostly situated in the interior, only 6701 deaths from cholera occurred, or 1·7 per 1000 inhabitants; while the 85 highest districts remained altogether untouched by the disease. The figures were similar in the epidemic of 1853-54; in those 136 districts, the mortality from cholera wos 16,295 (or 2 in 1000 inhabitants), and in the rest it was only 3802 (or 0·4 per 1000).[1] The researches instituted in France with a similar object have led to a very interesting result.[2] In the four epidemics of cholera which attained to general prevalence in that country (1832, 1849, 1853-54, and 1865-66), nine departments escaped the pestilence altogether (Cantal, Corrèze, Creuse, Dordogne, Gers, Landes, Lot, Lozére, Haut-Pyrénées), in two only scattered cases occurred in 1854 (Basses-Pyrénées and Tarn-et-Garonne); and all these, as well as seven others which were each visited only once, belong to the most elevated districts (over 600 metrès or 2000 feet). On the other hand, twelve departments, which count among the least elevated districts of France (Bouches-du-Rhône, Cher, Finistère, Loire-inférieure, Manche, Morbihan, Moselle, Nord, Oise, Seine, Somme), suffered in all the four epidemics. Further, as Fourcault has shown, and after him Valat,[3] the diffusion of cholera in France previous to 1849 was limited almost exclusively to the departments situated in the plains or valleys; when, in later epidemics, the disease extended to the mountainous departments (Vosges, Puy-de-Dôme, Eure, &c.), it was properly epidemic in the valleys only.[4]

Finally, if we take the several quarters of an affected place, situated at various heights, this dependence of cholera-diffusion upon elevation is proved by a truly overwhelming mass of recorded data. Everywhere and in almost every epidemic, it was the low-lying streets and parts of the town in which the disease commonly took origin and found its widest diffusion; and when it afterwards spread from these to more elevated points, it failed to reach the extent that it had in its primary

[1] On this subject, cf. Farr, 'Annual Report of the Registrar-General,' 1852, app., p. 61, 1856, app., p. 74.

[2] Scoutetten, in the 'Gaz. Hebd. de Méd.,' 1869, No. 53, p. 837.

[3] 'Bull. de l'Acad. de Med.,' xiv, No. 21.

[4] Cf. Jacquot, in the 'Gaz. méd. de Paris,' 1854, p. 529; Tholozan, ib., 1855, p. 435; Fortin, in the 'Bull. de l'Acad. de Méd.,' 1866-67, xxxii, p. 1226.

seats. Very often, indeed, the higher parts of the town or district were altogether exempt; so that Foureault is to a certain extent justified in formulating, for towns that are built like an amphitheatre or on a sloping surface, a law of cholera diffusion, in terms of which we distinguish three zones—one situated lowest, which is most severely attacked, an intermediate one with moderate amount of sickness, and an upper zone almost or altogether free from cholera. A particular interest attaches to the information from which we get the numerical data for this generalisation. Thus Richter[1] and Baer[2] found that in the Königsberg epidemic of 1831, the number of cases in the northern or low-lying part of the town was 37·3 per 1000 of the population, and in the southern or elevated part only 18·4. The sickness and mortality in the Hamburg epidemic of 1832, as recorded by Rothenburg,[3] was made up as follows:

	Sickness.	Mortality.
In the population of the whole town . . .	2·26 %	1·12 %.
„ „ low lying parts . . .	7·67 „	3·06 „
„ „ „ to the south	3·63 „	1·85 „
„ „ elevated parts to the east	1·97 „	1·04 „
„ „ „ „ west	1·25 „	0·65 „

In the Paris epidemic of 1832, the mortality from cholera amounted to 18·55 per thousand inhabitants in the most elevated quarter (17·30 mètres or 57 feet above the level of the Seine), and in the quarter lying lowest (avg. 3 mètres or 10 feet above the Seine), to 23·6.[4] Instructive also, for the question before us, are the mortality-statistics of the three London epidemics of cholera, as tabulated according to the elevation of the several quarters of the city.[5]

Table of deaths from cholera in London per 1000 *inhabitants, according to elevation of districts.*

At an elevation, above the level of the Thames, of	1848-49.	1853-54.	1866	
			Whole of London.	Eastern and North-Eastern Districts.
80 feet and upwards . . .	1·5	1·3	0·4	0·
60—80 feet	2·5	2·7	0·6	0·4
40—60 „	4·4	1·6	2·9	1·7
20—40 „	6·2	3·3	3·0	7·6
10—20 „	6·0	5·0	5·5	8·8
3—10 „	8·9	9·4	1·9	8·9
3 feet or less . . .	14·5	10·7	0·6	16·7

[1] In the 'Cholera Archiv,' iii, p. 183.

[2] 'Verhandl. der phys.-med. Gesellschaft in Königsberg über Cholera,' Bd. i, Heft 3.

[3] 'Die Cholera-Epidemie im J. 1832 in Hamburg,' Hamb., 1836.

[4] Boudin, 'Essai de géogr. médicale,' Paris, 1843, p. 32.

[5] Cf. Farr, in the 'Report of the Registrar-General,' 1852, App. p. 61, and

With reference to the mortality of 1866, it has to be kept in mind that the disease was principally confined to the eastern and north-eastern districts, and that of the 5548 fatal cases of cholera in that year in the whole metropolis, 4000 occurred in those two districts alone; so that, on this occasion also, the law which Farr deduced from the two former epidemics was fully borne out—the law, namely, that *the proportion of deaths among the inhabitants from cholera is inversely as the elevation of the ground.* The sickness and mortality in the three Oxford epidemics of 1832, 1849, and 1854, were, according to Acland,[1] respectively 3·3 and 1·8 per 1000 inhabitants in the higher parts of the town (*i.e.* more than a mean height of 30 feet above the water level), and, on the other hand, 9·8 and 5·3 respectively in the low-lying parts.

§ 108. Follows the Rivers.

It is almost unnecessary to say that differences of level in the several quarters of a locality do not *of themselves* exert an influence on the diffusion of cholera. This is quite obvious from the fact that exceptions on both sides occur not unfrequently; in districts of considerable extent, where the surface is generally hilly, as well as in localities within which there are great differences in level, the disease has either spread uniformly over the whole ground, or it may even have spared the low-lying parts to rage as an epidemic on the more elevated.

Thus, in the epidemic of 1834-35 at Marseilles, it was chiefly the elevated parts of the city that suffered; at Paris in 1853 it was on the hilly ground of the 11th and 12th arrondissements that most of the sickness occurred;[1] in the Prague epidemics of 1849 and 1866 the disease was as widely spread, and sometimes even more widely, at many very elevated points, as in the situations along the banks of the Moldau;[3] at Vienna in 1854 the elevated parts of the city suffered more than the low-lying; at Constantinople in 1847-48 no considerable differences could be ascertained in the amount of sickness at various heights within the city;[4] in Jamaica in 1851 the marshy tracts of coast and river-bank were visited by the disease less than the high-lying parts of the island.[5]

'Report on the Cholera Epidemic of 1866,' London, 1868; Radcliffe, in the 'Report of the Medical Officer of the Privy Council for 1866,' p. 291.

[1] 'Memoir on the Cholera at Oxford,' London, 1856, p. 49.

[2] Cf. 'Gaz. hebdom. de Méd.,' 1854, Nr. 14, 198.

[3] Pribram and Robitschek, in the 'Prager Viertelj. f. Heilk.,' 1868, i, 162.

[4] Rigler, in the 'Zeitschrift der Wiener Aerzte,' 1849, ii, p. 305.

[5] Perkin, 'Report of the Epid. Cholera in Jamaica,' London, 1852.

This dependence of the spread of cholera upon elevation must be ascribed, therefore, to certain peculiarities of soil which are met with, if not exclusively, yet for the most part, at low-lying points; and those peculiarities we may look for, practically, in *the more copious saturation of the ground, coupled with retention of organic matters undergoing decomposition.* One of the best proofs of this is the circumstance that *the diffusion of cholera mainly follows the course of rivers,* and that the amount of sickness diminishes in proportion as the disease in its progress travels farther from the margin of the river basin.

This point attracted attention in the first general outbreak of cholera in India in 1817.

"The tendency of the disease," says Jameson,[1] "to follow the course of rivers was so marked that it was impossible for anyone to regard it as a mere accident. Everywhere, from the source of the disease on the banks of the Ganges and Brahmaputra, to where it reaches the debouchments of the Nerbudda and Tapti [into the Arabian Sea], this mode of cholera-diffusion has struck the medical observer with astonishment. In the district of Bhagulpore the malady was so closely associated with the river banks that the poison hardly penetrated at all into the interior of the country, whereas the lowlands on the Ganges were almost depopulated." Orton also has expressed himself to the same effect.[2] As the cholera in Bengal followed the course of the Jumna and Ganges in its diffusion, so in Sind it followed the Indus.[3] In the Presidency of Madras, according to a statement of Balfour's,[4] there were within forty-eight years (1821-68) 152 outbreaks of cholera among troops on the march, the outbreak having occurred 102 times while the troops were marching along a river bank or in the immediate neighbourhood of one (within a distance of three miles). The diffusion of cholera in Russia in 1830 and 1831 chiefly followed the great rivers; likewise in 1831, and in all subsequent epidemics, it followed the Vistula and its tributaries in West Prussia, and in Silesia the course of the Oder and of smaller non-navigable streams such as the Hotzenplotz and Oppa.[5] In the Bohemian epidemic of 1832 the disease in many

[1] Loc. cit., p. 105.

[2] 'Essay on the Epidemic Cholera of India,' Madras, 1820, p. 410.

[3] Postans, 'Personal Observations on Sindh.,' London, 1843; Arnott, in the 'Trans. Bomb. Med. Soc.,' 1855, n. ser., ii, p. 175.

[4] 'Statistics of Cholera,' Madras, 1870.

[5] "Just as in 1831," says Brauser ('Die Cholera-Epidemie des Jahres 1852 in Preussen,' Berl., 1854, p. 2), "the spread of cholera in 1852 was unmistakably along the basins of rivers." In the report on the cholera epidemic in North Germany in 1873 ('Berichte der Cholera-Commission für das deutsche Reich.,' Berlin, 1879, vi, p. 301) I remark: "Evidence was adduced of the

parts remained limited to the banks of small streams.[1] In the epidemic of 1854 in Bavaria the places which suffered a true epidemic attack grouped themselves along the valleys or on the level margins of rivers and brooks.[2] In Würtemberg the disease was strictly confined to the country along the Enz;[3] and in Rhenish territory the cholera ran along the valley of the Rhine without spreading to either side. A glance at the map appended by Suerman to his history of the cholera epidemics in the Netherlands in 1832-34, conclusively shows that the disease in that country was confined almost exclusively to the banks of the rivers. Puytermans[4] reports from Belgium that the communes of the Canton Contich, situated on the banks of the Scheldt and Rupel, suffered the greatest ravages during the pestilence of 1832, while the inhabitants of the interior remained almost exempt. The one constant character, says Berg,[5] that belonged to all the localities in Sweden attacked by cholera in 1850 was, that they were all either in the neighbourhood of a lake or situated on the banks of a lake or river. With reference to the diffusion of cholera in France in 1832, we read: "The departments with water on one or more sides, those, namely, situated on the coast or at the confluence of several rivers, have, *ceteris paribus*, suffered more harm than the more elevated;"[6] the same fact was noted in the epidemic of 1848-49, according to Vallat, and in the epidemic of 1853-54, according to the reports of Destrem[7] and Jacquot.[8] In the Danubian Principalities the diffusion of cholera in 1848 and 1849 chiefly followed the Danube and its tributaries;[9] and the same fact was observed when the disease broke out in Canada and the United States, both at the first epidemic and subsequently;[10] in the cholera epidemic of 1850 in Granada, where the disease followed the course of the Rio Magdalena;[11] in the Mauritius in 1854,[12] and in other places.

circumstance that in this, as in almost all former epidemics of cholera in Germany, the disease was associated in a remarkable way with the river traffic (*i. e.* the river banks), that it was chiefly confined to the riparian zone, and that it became prevalent in districts lying at a distance from the stream only at a later period, if it did not spare those districts altogether."

[1] Report in the 'Cholera-Archiv,' Berlin, 1832, ii, p. 272.

[2] 'Haupt-Bericht über die Cholera-Epidemie des Jahres 1854 in Bayern,' München, 1857, pp. 310 ff, 807, No. 13.

[3] Keyler, in the 'Würt. med. Corrspdzbl.,' 1849, xix, p. 210.

[4] 'Archives de la méd. Belge,' 1845, Aug., p. 181.

[5] 'Repporter om Cholerafarsoten i Sverige år, 1850,' Stockh., 1851.

'Gaz. méd. de Paris,' 1832, p. 410. [7] 'Gaz. des hôpit.,' 1854, No. 105.

[8] 'Gaz. méd. de Paris,' 1854, No. 35, p. 529. [9] Barasch.

[10] The spread of the disease in 1850 along the banks of the Sacramento in California is vouched for by Logan ('Southern Med. Reports,' New Orleans, 1851, ii, 463), and confirmed by Parslow (l. c.); Farnsworth ('Philad. Med. and Surg. Reporter,' 1870, Mar. 26), in like manner, mentions that it spread in 1866 along the river valleys in Iowa.

[11] Wis, l. c.

[12] Clerihew, 'Med. Times and Gaz.,' 1856, Aug. 9.

That this relatively close connexion between cholera and the river banks ought not to be set down to the disease spreading by means of the traffic of the water highways, may be deduced from the fact that the connexion holds good as much for the smaller streams and brooks as for the large or navigable watercourses. It further appears that we are not concerned here with a diffusion of the morbid poison by infected water used for drinking or cooking; for the progress of the disease has been as often up-stream as down-stream. The copious soaking of the ground, owing to its situation, is undoubtedly the real cause. And that view is otherwise confirmed: firstly, by the fact that the saturation of the soil by atmospheric precipitations is not without influence on the epidemic occurrence and prevalence of cholera; and, secondly, by the fact, which experience has proved over and over again, that in the affected localities the seats of the disease have been chiefly the low-lying quarters with a moist soil abounding in decomposing organic matters, and that, under those circumstances, the epidemic has been often limited to particular streets or groups of houses, and even to particular houses.

The earliest epidemics of cholera on Indian soil supplied observations of that kind, such as those of Scott from Guntoor, of Gravier from Pondicherry,[1] and others; Parkes observes that the epidemic of 1842 at Moulmein (British Burmah) was exclusively in the streets close to the banks of the river Salwen.[2] Observations to the same effect were made in various towns of Russia during the first cholera epidemic, for example, in Odessa, Moscow,[3] and Warsaw;[4] and they were confirmed in the subsequent epidemics.[5] In Müller's report of the Riga epidemic of 1848 it is stated:[6] "In the more airy and spacious suburbs, cases of sickness were met with most frequently in particular streets, which were either in a filthy state or were situated near a piece of stagnant water; . . . the disease showed itself in greatest intensity in a street built along a stagnant backwater of the Düna, and consisting of inferior houses mostly tenanted by the poor." With reference to the epidemic at Dorpat in 1871, Weyrich remarks:[7] "As regards the spread

[1] 'Annal. de la med. physiol.,' xi, 269.

[2] 'Researches on the Pathology and Treatment of Asiatic Cholera,' London, 1847.

[3] Jänichen, in the 'Gaz. méd. de Paris,' 1831, March.

[4] Raciborsky, in the 'Gaz. des hôpit.,' 1837, No. 139.

[5] Cf. Frettenbacher, l. c.

[6] 'Beiträge zur Heilkunde, herausgegeben von Rigaer Aerzten, 1849,' i, p. 31.

[7] 'Dorpater med. Zeitschrift,' 1873, iv.

of the epidemic in the town itself, it soon became clear that it had chosen certain spots in the river valley for its proper sphere, the more elevated quarters of the town having no epidemic, but only a few scattered cases." In the numerous epidemics of cholera which have been prevalent in Königsberg, the low-lying and damp parts of the town have suffered most;[1] in the epidemics at Danzig from 1849 to 1867, as appears from the inquiries of Liévin,[2] the cross streets situated nearest to the Motlau suffered more than streets running parallel to the water at some distance from it, and the filthy part of the town, which is traversed and in part surrounded by the Radaune and its canals, contributed by far the largest quota to the total sickness. Similar observations occur for the epidemics of cholera in Lübeck,[3] Hamburg,[4] many places in Holstein[5] and Mecklenburg,[6] in Lüneburg,[7] Anklam,[8] Stettin,[9] and Berlin.[10] The same conclusion is deducible from the inquiries on the spread of cholera in 1854 in the various places which the disease visited in Bavaria,[11] and from the records of the epidemics at Ingolstadt in 1854 and 1873,[12] and of the various outbreaks at Prague,[13] Vienna, and other places.[14] The official reports of epidemics of cholera in Sweden[15] dwell almost always on the damp situation of the localities attacked. As regards the import of the facts in England,

[1] Cf. Möller, in the 'Berl. klin. Wochenschrift,' 1866, p. 471; Schieferdecker, 'Die Cholera-Epidemie vom Jahre 1871 in Königsberg,' Königsb., 1873, p. 17.

[2] 'Danzig und die Cholera, &c.,' Danzig, 1869, p. 26.

[3] Martini, in the 'Allgemein. med. Ztg.,' 1832, p. 1256; Cordes, in the 'Zeitschr. f. Biologie,' 1868, iv, 167.

[4] Rothenburg, in the (Hamb.) 'Ztschr. f. d. ges. Med.,' 1836, ii, 425; Warburg, ib., 1838, ix, 9.

[5] Pfaff, l. c., 65.

[6] Spitta, 'Die asiatische Cholera in Grossh. Mecklenburg-Schwerin,' Rostock, 1832.

[7] Münchmeyer, in the (Hamb.) 'Mag. des ges. Heilkunde,' 1832, xxiii, 252.

[8] Ziegler, 'Die Cholera nach einer einfachen Methode zu überwinden,' Anklam, 1869.

[9] Goeden, in the 'Berl. klin. Wochenschrift,' 1872, No. 33.

[10] Breyer, in 'Hufel. Journ.,' lxxiv, pt. i, p. 3; Schütz, in 'Virchow's Archiv,' 1849, ii, 380.

[11] 'Generalbericht,' p. 809.

[12] Mair, in the 'Bair. ärztl. Intelligenzbl.,' 1874, No. 31.

[13] Krombholz, 'General-Rapport über die Cholera zu Prag,' Prag, 1836, p. 32; Loeschner, 'Schluss-bericht über die Cholera-epidemie, 1849-51 in Prag,' Prag, 1854; Popper, in the 'Allg. Zeitschr. für Epidemiologie,' 1876, ii, 285 ("as regards the area of distribution in the Prague epidemics, it is mostly the lower parts of the town near the Moldau that have been found to be the centres of disease").

[14] For the corresponding facts in the North German epidemic of 1873, cf. Hirsch, 'Bericht der Cholera-Commission für das deutsche Reich,' 1879, vi, 305, *et seq.*

[15] 'Sundhets-Collegii Berättelse.'

Radcliffe[1] remarks: "In all epidemics of cholera in this country, the state of the soil and the degree to which it was charged with moisture and decomposing organic matter, especially excrementitious, has been held to exercise an important influence over the localisation of the disease." Gendrin[2] says: "The chief general predisposing cause of cholera, about which authors are agreed, and which our study of the Paris epidemic has quite confirmed, is the crowding of the inhabitants along the river banks;" and as evidence of this he gives statistics of the fatal cases in the several arrondissements. These experiences were repeated in Paris in 1849,[3] as well as at Calais, Rouen,[4] Corbeil,[5] and many of the places affected in Southern France,[6] and in numerous towns of Italy and Spain. The same thing occurred in the Western Hemisphere. In New York the disease was on every occasion most prevalent in the low-lying, damp, and filthy streets;[7] so also in Philadelphia, where the dry and clean western quarters of the city (Penn District, Spring Garden, Northern Liberties) were almost exempt;[8] and in Pittsburg[9] and Hartford[10] (Conn.) in 1854, and New Orleans in 1873.[11]

§ 109. Favouring Characters of the Soil.

In all probability the active pathogenic factor in these conditions of soil is to be sought for in the processes of decomposition which the organic substances, in or upon the ground, undergo under the circumstances mentioned. It is

[1] 'Ninth Report of the Medical Officer of the Privy Council, 1866,' London, 1867, 292. Compare also the reports of Greenhow on the epidemics at Tynemouth from 1832 to 1853 in the 'Transact. of the Epidemiol. Soc.,' i; 'Journal of Public Health,' p. 25; of Adams for Glasgow ('Edin. Med. and Surg. Journ.,' 1849, Oct., p. 286); of Acland (l. c., p. 46) for Oxford; of Sutherland for London ('Report on Epidemic Cholera,' Lond., 1853, p. 27); of Craigie for Newhaven ('Edin. Med. and Surg. Journ.,' 1832, April, p. 339); and of Proudfoot for Kendal (ib., 1833, Jan., p. 75).

[2] 'Transactions medicales,' 1832, viii, 395.

[3] Report in the 'Arch. gen. de méd.,' 1849, Dec., p. 501.

[4] Hellis, 'Souvenirs du Cholera à Rouen,' Paris, 1833.

[5] Report in the 'Gaz. méd. de Paris,' 1832, p. 252.

[6] Hergt, l. c., 20; Arnaud, in the 'Revue thérap. du midi,' 1855, Oct.

[7] Reese, 'Treat. on Epid. Cholera,' New York, 1833, p. 31; Bowron, 'Observ. on Malignant Cholera,' New York, 1835, p. 22; Hutchinson, in the 'New York Journ. of Med.,' 1835, Jan.

[8] 'Statistics of Cholera,' Philad., 1849.

[9] Gallaher, Pollock, and Draine, in the 'Amer. Journ. of Med. Sc.,' 1855, Oct., p. 335.

[10] Russel, in the 'Proc. of the Connecticut State Med. Soc.,' 1855.

[11] Jones, in the 'Boston Med. and Surg. Journ.,' 1873, July.

on that hypothesis that we can understand how *the drying of the soil after it has been soaked* (whether by direct atmospheric precipitations, or by fluctuations in the level of the subsoil water more or less closely connected with them) has proved to be particularly favorable to the development of cholera epidemics, as we shall see when we come to speak of the influence exerted by states of the weather. Again, the disease has attained its widest diffusion and its greatest intensity in those localities or districts distinguished by a certain *physical character of the soil*, viz. permeability to water and air, and a *certain kind of rock*, namely, rock with a capacity for retaining the moisture that has sunk in; and these, together with a certain amount of organic detritus, correspond very closely to the above-mentioned conditions for the diffusion of cholera.

At the first general outbreak of cholera on Indian soil, the attention of several observers was drawn to the fact that localities with *a rocky bottom* enjoyed a relative if not an absolute immunity from the disease, and that the removal of troops from the alluvial plains where the pestilence raged was followed by its speedy extinction among them. Similar observations were afterwards made at several places in Russia as well as in Canada in 1832,[1] and in Nova Scotia in 1834.[2] The merit of stating in general terms, as well as of illustrating in detail, this dependence of the diffusion of cholera upon geological and physical characters of the soil, belongs to Boubée,[3] who showed, in a communication addressed to the Académie des Sciences, that the disease on its first appearance on European soil spread with greatest rapidity and with widest sweep over tracts of country that had an alluvial or tertiary bottom; that, on the other hand, it made very slow progress over soils belonging to the older formations, especially the primary rocks, on which it lost its malignant character, and quickly died out. At the same time he gave it as his opinion that it was not the geological character of the soil *in itself*, but the saturation dependent thereon, in which the

[1] 'Statist. Reports on the Sickness among the Troops in the United Kingdom, &c.,' London, 1839, 30 *b*.

[2] Ib., 18 *b*.

[3] 'Revue méd.,' 1832, Août, p. 311.

true explanation of that phenomenon was to be looked for, and he opined that cholera might very well occur on a rocky bottom, wherever the latter was disintegrated by the weather, or broken up, or covered with detritus, and so made liable to become sodden like the permeable rock of a newer formation. This question was investigated anew, on the second outbreak of the disease, by Fourcault, who had now a larger body of materials at his command; and, so far as concerned France, the conclusion of Boubée was upheld. Among kinds of soil, he says,[1] that are favorable to the diffusion of cholera, alluvium takes the first rank, and next follow coarse-grained limestone, clay, coal-bearing formations, and mountain limestone; more rarely an epidemic diffusion of cholera occurs upon firm sandstone, flint conglomerate, and chalk; but, upon transition formations and primary rock, it occurs only when they have become saturated in consequence of cleavage. Boubée[2] found a further confirmation of his earlier observations in the mode of diffusion of the cholera in the Pyrenees during the epidemic of 1854; all the localities with a granite bottom were spared, but that immunity ceased wherever a layer of alluvium or debris, if it were only a thin one, overlaid the rock. In the same epidemic, Dechambre[3] noticed the relative immunity enjoyed by the western parts of France (Bretagne, Poitou, &c.) which rested on primary rocks, as well as by the districts belonging to the secondary formations, including the districts from Ploërmel to Chateau Neuf and from Laval to Angers. Vidal[4] showed that the department of the Loire, with the granite rock of the Pital and the Forez mountains, had been always exempt from cholera, as well as the volcanic soil of Auvergne and the rocky mountainous district of Morwan; whereas the disease had attained a wide diffusion on the tertiary and alluvial soil of the Gironde and of the Rhone delta. Pettenkofer came to the same conclusion in his very thorough researches on the spreading of cholera in Bavaria in 1854. "All the

[1] 'Gaz. méd. de Paris,' 1849, pp. 338, 357. Compare also his researches on the epidemic of 1834-35 in Southern France, ib., 1850, pp. 373, 411.

[2] Ib., 1854, pp. 634, 680.

[3] 'Gaz. hebdom. de méd.,' 1854, No. 62, 1855, No. 27.

[4] Ib., 1854, No. 65.

localities, or subdivisions of them, attacked by epidemic cholera," he says in his summary,[1] "rest upon porous soil permeable to water and air . . . So far as relates to localities or particular spots, resting directly upon compact stone or upon rock, not permeated by water, we have observed in them for the most part no cases of cholera, sometimes a few sporadic cases, but never an epidemic." And he arrived at the same result for the epidemic of 1865 in Altenburg,[2] and for that of 1866 in several localities of Thuriginia.[3] To the same effect are the reports upon the diffusion of cholera in the epidemic of 1855, through the Hungarian counties of Sohl and Honth,[4] and in the Luxemburg epidemics of 1865 and 1866;[5] and again in the epidemic at Olmütz in 1866,[6] and in the Würtemburg epidemics from 1849 to 1873.[7]

All the epidemics that have hitherto occurred in the Netherlands go to prove by details, that can be reduced to the most interesting degree of exactness and shown upon the map,[8] that the disease attained a proper epidemic diffusion only in those districts which had an alluvial soil; so that the margin of the alluvial formation, where it abuts on the diluvial, makes a sharp line of demarcation between the infected and the exempted districts.

Another circumstance worthy of note is that even a permeable soil, if it be unable from its physical or geological qualities to retain its imbibed moisture, as well as a soil completely swamped and permanently under water, is little favorable to the epidemic diffusion of cholera. Thus we read in the report on the cholera epidemic of 1873 in North Germany, with reference to the departments of Bromberg, Posen, Marienwerder, and Breslau:[9] "those

[1] 'Hauptbericht über die Cholera-Epidemie des J. 1854 im Konigr. Bayern.,' Munich, 1857, p. 807.

[2] 'Zeitschrift für Biologie,' 1866, ii, p. 82.

[3] 'Bayer ärztl. Intelligenzbl.,' 1867, No. 9, p. 124.

[4] Salawa, in the 'Ungar. Zeitschrift für Natur- und Heilkunde,' 1856, p. 301.

[5] 'Bull. de la soc. des sc. méd. du Grand Duché de Luxembourg,' 1868, p. 210.

[6] Pissling, in the 'Wiener med. Wochenschrift,' 1866, No. 86.

[7] Burkart, in the 'Zeitschrift für Biologie,' 1876, xii, p. 400.

[8] 'De Cholera-Epidemie in Nederland in 1866 en 1867,' s'Gravenhage, 1875, p. 383.

[9] 'VI Bericht der Cholera-Commission für das deutsche Reich,' Berlin, 1879 p. 305.

districts whose surface soil was formed by a more or less considerable stratum of heavy loam containing sand and mould, furnished the proper seat of the epidemic, while the highly permeable sandy bottom of the so-called 'stream country' was almost entirely free from the disease—a fact all the more noteworthy as it shows the circumstances of the year 1873 to have been the same as in all previous cholera years, according to what we are told by the various observers in those localities." Similar observations have been communicated from India[1] and elsewhere.

There are not wanting, however, published reports which endeavour to show that cholera may diffuse itself epidemically upon a rocky and hard bottom as well; we find examples of this in the occurrence of the disease on the limestone rocks of Malta, on the "Karst" of Carniola,[2] on the granite soil of Helsingfors,[3] and on the lateritious soils in Hindostan. But most of these alleged instances are based, as exact inquiries have shown, upon erroneous assumptions or imperfect research. Thus, it has been shown by Pettenkofer that the limestone rock of Malta is a very soft and porous kind of stone, which sucks up moisture like a sponge,[4] and that the localities in the "Karst" region that have suffered from cholera are not those situated upon impervious (limestone) rock—these having in fact remained quite untouched by the disease—but those with a stratum of loamy soil over the rocks, or filling up the broad and deep fissures and gaps among them.[5] The statement that the high-lying part of the town of Helsingfors rests on the bare rock has been rejected as erroneous by Quist, who says that there is a layer of alluvium lying upon the granite.[6] In their latest papers on the cholera in India, Lewis and Cunningham corroborate the fact already made out by earlier observers (Macpherson, Macnamara, Cornish, and others), that the lateritious soil of that country (ferruginous clay) upon which severe epidemics

[1] McLelland, in the 'Annals of Military and Naval Surgery,' 1863, i, p. 207.
[2] Drasche, 'Die epidemische Cholera,' Vienna, 1860, p. 156.
[3] Nylander, in the 'Finska Läkare-Sällskapets Handlingar,' 1850, iv, p. 199.
[4] 'Zeitschrift für Biologie,' 1870, vi, p. 143.
[5] 'Bayer. ärztl. Intelligenzbl.,' 1861, p. 89.
[6] 'Om Koleran i Helsingfors, 1871, &c.,' Helsingf., 1872, p. 81.

of cholera have raged, is highly porous.[1] In like manner we might explain away other instances of cholera said to have occurred epidemically on hard rock, by regarding them from the point of view, *not of the mineralogical but of the physical character of the soil, and not of the formation and kind of rock which pertains to a district in general, but of the immediate foundations of the inhabited places therein.*

§ 110. Influence of Seasons and Weather.

The epidemic spreading of cholera appears to depend not less upon *atmospheric influences* than upon the conditions of soil just spoken of; and that dependence is shown in the connexion between cholera and particular *seasons of the year* and *states of the weather*. The following tables furnish particulars of the time of prevalence of the disease at various parts of *India* unlike in their climate and soil:

Table of the Cholera Mortality, the Temperature, and the Rainfall (India I).

	Calcutta.			Bombay.		
	No. of deaths from cholera.[2]	Mean temp. (Fahr.)	Rainfall (in inches).	No. of deaths from cholera.[3]	Mean temp. (Fahr.)	Rainfall (in inches).
January	9,105	67·7	0·44	3296	74	—
February	12,572	73·0	0·83	2729	76	—
March	19,558	80·5	1·28	3270	80	—
April	24,040	84·7	2·49	4032	83	—
May	16,641	86·2	5·46	3784	86	0·5
June	8,556	84·9	12·13	3972	83	22·7
July	5,297	83·5	12·64	2312	81	24·5
August	5,124	83·1	13·71	1339	81	12·4
September	5,478	83·1	10·17	857	80	10·6
October	8,016	81·5	5·61	1118	82	1·7
November	11,112	74·9	0·66	1411	79	0·3
December	10,334	68·1	0·24	2633	76	—

[1] 'Cholera in Relation to certain Physical Phenomena,' Calcutta, 1878, p. 51.

[2] Twenty-six years' observations, within the period from 1830 to 1860, compiled by Macpherson ('Cholera in its Home,' London, 1866), and observations for the years 1865-76, published by Lewis and Cunningham ('Cholera in Relation to certain Physical Phenomena,' p. 8).

[3] Fourteen years' observations by Leith, published by Macpherson in the 'Med. Times and Gaz.,' 1867, Nov. 29, p. 562.

Table of the Cholera Mortality, the Temperature, and the Rainfall (India II).

	Madras.			North-West Provinces.					Deccan.
	Deaths from cholera.[1]	Mean temp. (Fahr.).	Rainfall (in inches).	Deaths in prisons.[2]	Deaths among troops and prisoners at 25 stations[3]	Agra. — Deaths among soldiers and prisoners.[4]	Mean temp. (Fahr.).	Rainfall (in inches).	Deaths from cholera in the State of Hyderabad.[5]
January . .	2226	74·5	1·01	8	124	—	57	1·2	177
February .	2541	76·3	0·32	8	132	1	63	1·1	56
March . .	1580	79·9	0·31	24	720	2	74	—	79
April . . .	854	83·5	0·81	31	1382	3	85	0·2	140
May . . .	880	85·8	2·92	54	1902	6	90	0·7	604
June . . .	712	85·8	2·21	114	2778	236	95	0·3	425
July . . .	1774	84·1	2·41	222	4778	234	87	9·8	1063
August . .	1802	83·1	3·86	268	8676	314	85	10·0	1634
September .	1986	82·5	4·28	114	3231	55	84	4·0	152
October . .	1675	79·9	12·95	78	942	3	75	0·6	25
November .	1220	76·8	11·80	69	470	4	69	—	128
December .	1183	74·9	6·31	10	203	—	58	—	1680[6]

It appears from these tables that the season of cholera prevalence (as estimated by the number of fatal cases) is very different in the different parts of India. In Calcutta (and in Bengal generally), the mean falls in the month of November, with 11,112 out of 135,833 deaths; from that point the numbers diminish somewhat in the two following months; in February a considerable rise in the deaths begins, reaching its maximum in April; in May an abatement sets in, which continues through June and July; the minimum is in August, and from then onwards there is a rise which reaches its highest point in November. In four months (February to May) the cholera mortality exceeds the mean for the year, in three

[1] Observations for the years 1855-64, given by Cornish in the 'Med. Times and Gaz.,' 1868, March 21, p. 312.

[2] Observations for the years 1844-53, given by Lawson in the 'Army Medical Reports for 1866,' p. 385. The figures give the proportion for each month in 1000 annual deaths from cholera.

[3] Observations for four years, given by Lewis and Cunningham, l. c., p. 73.

[4] Observations for the years 1857-65, given by Macpherson in the 'Med. Times and Gaz.,' 1867, Nov. 23, p. 563.

[5] Observations made at thirty-two towns and military stations within the period from 1857 to 1869, given by Balfour in the 'Madras Monthly Journ. Med. Sc.,' 1870, i, p. 210.

[6] The high December mortality was due to a severe epidemic (1474 deaths) 1868 and 1869 in the city of Hyderabad.

months (November to January) it stands at the mean, and in five months (June to October) it falls more or less below the mean. The largest number of deaths occur in April, the smallest number in August. As regards the progression of cholera in Bombay, the figures come out in somewhat the same order. Here the mean occurs in December, after which there begins a rise that also reaches its highest point in April; then follows a gradual sinking, so that the number of deaths in July again touches the mean of the year; a further sinking conducts to the minimum in September, and from that point there is a rise until the mean is reached in December. In this Presidency the amount of sickness exceeds the mean of the year in six months (January to June), stands at the mean in two months (July and December), and falls below it in four months (August to November). In Madras the distribution of the disease throughout the year is materially different; here the maximum occurs in January and February, in March the mortality sinks to the mean of the year, and then still farther until it touches the lowest point in June; it then rises to a second maximum in September, after which there is an abatement until December, and thereafter a sudden rise in January. Here there are two maxima, one in February and another in September, with the minimum in June. Finally, in the North-West Provinces, we have to distinguish two periods, one embracing seven months from April to October, with cholera prevalent as an epidemic, and the other, from November to March, in which the disease occurs in more isolated cases. Here the maximum falls in the months of July and August, the minimum in January and February. On the high table-land of the Deccan, the amount of sickness is distributed over the year in almost the same way; and thus we find that the amount of cholera in those two regions, in the several seasons of the year, is almost in the same proportion as in sub-tropical and temperate latitudes outside India. I shall endeavour in the sequel to show what factors are concerned in these variations in the amount of cholera with respect to the various seasons of the year. In the meantime I adduce the results of an investigation (embracing 920 epidemics) of the time of the appearance or prevalence of cholera in extra-Indian countries in the several months:

Table of Cholera Epidemics in Extra-Indian Countries in the several Months.

Locality of observation in the isothermal zones.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.
20° C. [68° Fahr.] and over .	—	5	7	10	22	11	19	9	4	7	7	9
From 15—20° C. [58°—68° F.]	2	2	2	6	7	36	38	25	10	8	2	4
„ 10—15° C. [50°—58° F.]	3	1	8	18	20	52	68	58	18	12	7	6
„ 5—10° C. [41°—50° F.]	—	2	8	11	14	45	71	75	54	34	9	4
5° C. [41° Fahr.] and under .	1	—	1	1	1	7	20	15	12	8	1	3
	6	10	26	46	65	151	216	182	98	69	26	26
	42 Winter.			261 Spring.			496 Summer.			121 Autumn.		

From this it appears that the largest number of epidemics begin in the months from June to August, and the fewest in the months of January and February. An inquiry into the distribution of fatal cases over the several months of the year leads to almost the same result. Thus, in England, Prussia, Russia, and Tuscany, there was a mortality from cholera in the undermentioned epidemics as follows:

Table of Cholera Mortality in England. &c., in the several Months.

	Whole of England.				Lond.	Kingdom of Prussia.		Russia.			Tuscany
	1831.	1832.	1848.	1849.	1866.	1852.	1853.	1853.	1854.	1855.	1855.
January . .	—	614	—	658	—	—	450	949	229	606	—
February .	—	708	—	371	—	—	319	408	84	356	102
March . .	—	1519	—	302	—	—	141	786	120	409	49
April . . .	—	1401	—	107	—	—	14	1,516	239	482	51
May . . .	—	748	—	327	—	—	—	1,901	305	1,731	240
June . . .	—	1363	—	2,046	—	—	—	5,223	1274	20,930	681
July . . .	—	4816	—	7,570	1296	721	—	30,269	2764	52,554	4,837
August . .	—	8875	—	15,872	2555	8,314	—	40,318	6554	44,114	11,227
September .	—	5479	—	20,379	864	18,627	—	10,439	1715	7,735	7,065
October . .	—	4080	354	4,654	645	7,134	—	4,999	587	1,465	1,789
November .	97	802	376	844	180	3,137	—	1,766	376	590	—
December .	282	140	375	163	—	1,583	—	303	385	75	—

§ 111. Influence of Heat.

It is obvious that we must look for an explanation of this kind of dependence of cholera-diffusion on the season of the year, in the weather-influences peculiar to certain seasons, and more particularly in the influence of *temperature* and *atmospheric precipitations*. There can be no doubt that a relatively high *temperature* materially favours the production of cholera; the proof of this, however, will be found, not so much in the circumstance that the disease as an endemic or epidemic reaches the acme of its prevalence at the same time as the temperature touches its highest point, but rather (on the assumption of a general epidemic preponderance during high temperature) in the perfect correspondence between the abatement of the sickness and a considerable drop of the temperature, or, in higher latitudes, the correspondence between the extinction of the epidemic and the setting in of positively cold weather.

From the statistics given above of cholera in India, it follows that in Calcutta the curve begins to ascend in October with the setting in of a cooler temperature, and that the remission of the endemic corresponds with the arrival of the hot season in May; the circumstances in Bombay are perfectly similar; in Madras the minimum of sickness falls precisely in the hot season; in the North-West Provinces also, *i.e.* the central and northern parts of Hindostan, as well as in the Deccan, the highest point of sickness corresponds with the abatement of the highest temperature. In Java the cholera is prevalent mostly in the months from June to September, characterised by the coolness of the night and morning;[1] and in Anam (Cochin China and Cambodia) in the months from February to May, the time of the north-east monsoon.[2] In temperate latitudes, as the cholera statistics for England, Russia, and other countries show (2nd Table on p. 459), the disease becomes more decidedly prevalent at the time of greatest heat. There are not wanting even observations which would indicate that the epidemic keeps pace with the rise and fall of the temperature, both in range and severity. But in numerous other epidemics, as, for example, those of 1831-32 and 1866 in Prague, that of 1848 in Riga, 1850 in Holstein, 1853 in Edinburgh, 1855 and 1866 in Halle, 1867 in Zürich, and the various epidemics that have occurred at Lübeck and Helsingfors, it has not been possible to make out any such constant relation between the height of the tempe-

[1] Compare 'Arch. de méd. navale,' 1868, Dec., p. 408.

[2] Laure, 'Histoire méd. de la Marine Française, &c.,' Paris, 1864, p. 99. Richaud, in the 'Arch. de méd. navale,' 1864, May, p. 340. Thil, 'Remarques sur les principales maladies de la Cochin Chine,' Paris, 1866, p. 29.

rature and the severity of the epidemic; and still less can we trace any such relationship when one epidemic is compared with another with reference to their severity and to the temperature at the time.[1]

Much more obvious, as we have said, is the effect of a decrease of heat in abating or extinguishing the epidemic. Even in the North-West Provinces of India and in the Punjaub, this influence makes itself decidedly felt; "epidemics at that (the cold) season," says Macpherson,[2] referring to those provinces, "are unknown, and even sporadic cases are extremely rare;" and the conclusions of Gordon,[3] Macnamara,[4] and others are the same in effect. The same circumstance comes out still more prominently with respect to higher latitudes; with the approach of true winter cold, the sickness either disappears altogether, or, if it lasts through the winter, it mostly lays aside its proper epidemic character, and does not attain its wider diffusion before the spring, and, indeed, not usually until the beginning of summer. Classical examples of this are furnished by the behaviour of cholera in the winters of 1821-22 and 1822-23 in Persia, Mesopotamia, and Syria, of 1823 in Astrakhan,[5] 1829-30 in Persia, 1831-32 in Astrakhan, Moravia, and Galicia,[6] in Silesia,[7] on the north coast of Germany,[8] in Louisville, Cincinnati,[9] Baltimore,[10] and other places in the United States, 1832-33 and 1833-34 in Belgium,[11] 1834 to 1835 in the South of France, 1835-36 in Upper Italy, 1836-37 in Poland,[12] 1846-47 in Persia, 1847-48 in Russia and Turkey, 1848-49, in Belgium, many parts of Germany, at Glasgow, and in Norway,[13]

[1] Delbrück ('Zeitschrift für Biologie,' 1868, iv, p. 238) has expressed the belief that a special significance for the spread of cholera attaches to the *degree of temperature of the soil*. Pfeiffer (ib., 1871, vii, p. 262), comparing the number of deaths from cholera in Prussia in the years from 1848 to 1859 with the temperature of the ground at Brussels for the corresponding months, and the cholera mortality in England in 1832 and 1848-49 with the temperature of the ground at Edinburgh, found that a rise or fall of the temperature at a depth of one to two mètres (3 to 6½ feet) corresponded to increase or decrease of the epidemic. The results of a three years' inquiry in India directed to the point (see Lewis and Cunningham, l. c., p. 32) do not justify us, any more than the inquiries already referred to, in forming an opinion as to the importance of this factor in the etiology.

[2] 'Med. Times and Gaz.,' 1867, Nov. 23, p. 563.

[3] Ib., 1856, Oct.

[4] 'History of Cholera,' p. 448.

[5] Jähnichen, in 'Hecker's Wissensch. Annal.,' xix, p. 385.

[6] 'Oestr. med. Jahrb.,' n. s., iv, p. 552.

[7] 'Sanitätsbericht für Schlesien vom J. 1831,' p. 196.

[8] Pfaff, 'Mittheilungen,' 1st year, pt. i, p. 39.

[9] Harrison, in the 'Baltimore Med. and Surg. Journ.,' 1834, ii, p. 265.

[10] Sexton, in the 'North Amer. Arch. of Med.,' 1835, ii, p. 314.

[11] Meynne, 'Topogr. méd. de la Belgique,' Brussels, 1865, p. 232.

[12] Raciborsky, in the 'Gaz. des Hôpit.,' 1837, No. 139.

[13] Löberg, in the 'Norsk. Magaz. for Laegevidensk.,' 1849, n. s., iii, p. 271; Kjerulf, ib., p. 541.

1853-54 in London, 1854-55 in Hungary and France, 1865-66 in Roumania, 1872-73 in Bohemia,[1] and 1873-4 in the Prussian department of Oppeln. Severe epidemics of cholera in the winter time are among the rarest events: such were those of 1829 at Orenburg, 1830-31 at Moscow, 1831-32 at Prague, 1846 in Armenia, 1852-53 at St. Petersburg,[2] and of 1866 in the Hungarian county of Sohl.[3]

§ 112. Influence of Moisture—Pettenkofer's Law of the Sub-soil Water.

As to the influence of *atmospheric precipitations* upon the production and diffusion of cholera, we find much interesting light thrown upon the question by the almost constant relation that exists between the two in India. In Calcutta, as well as in all that part of the Bengal Presidency which belongs to the endemic cholera region of India, the maximum of sickness falls, as the table on p. 456 will show, in the dry season (October to May), and more particularly in that portion of the dry season which is at the same time the season of greatest heat (March to May). The circumstances in Bombay are very similar; here also an abatement of the disease occurs at the setting in of the rains (see Table), and an increase of the epidemic during the dry season (December to June), and here also the maximum of sickness falls in the hottest part of that section of the year (April to June). In Madras (Table, p. 457) one of the two maximum periods begins in January, *i.e.* at the end of the rainy season (October to December), and lasts until March; the other develops when a certain amount of moisture begins to fall in July, and lasts to the setting in of the heavy rains in October. Quite different from those of Calcutta and Bombay, are the circumstances of the North-West Provinces, where the highest point of the epidemic corresponds to the rainy season, *i.e.* the setting in of the south-west monsoon (July to September); in fact the epidemic in its development keeps pace with the monsoon in its course westwards, so that the Punjaub is usually the last province to be attacked. "No sooner do the rains cease,"

[1] Kaulich, in the 'Prager Vierteljahr. für Heilkd.,' 1875, ii, p. 95.

[2] Everard, in the 'Bull. de l'Acad. de Méd. de Belgique,' 1854, xiii, p. 201.

[3] Heinrich, in the 'Wien. med. Presse,' 1872, No. 50, p. 1158.

says Macnamara,[1] with reference to the epidemic prevalence of cholera in the North-West Provinces, "and the dry west winds of the upper provinces set in—at the latter end of September or beginning of October—than cholera begins to decline over the North-West." During the time from October to June often not a drop of rain falls in the North-West Provinces, and for just as long does the district remain, as a rule, free from cholera. The conclusion that follows from this, with respect to the influence of the fall of moisture on the occurrence of cholera in that region of Hindostan, finds considerable support in the circumstance that epidemic outbreaks of cholera occurring there at unusual seasons of the year are for the most part associated with sudden and heavy falls of rain,[2] and that, as in other parts of India, unusually copious rains set a limit to the spread of the epidemic, or may bring it even to an end.[3]

Less marked, but still recognisable, is the influence of the rainfall on the occurrence, prevalence, and disappearance of the disease in those extra-Indian countries where the circumstances of the weather have a less definite uniformity. In them the epidemic develops usually in connexion with the spring or summer rains, especially at their cessation, lasts through the warm season, and finally dies out when the rains begin in late autumn and colder weather sets in.[4] But

[1] 'History of Asiatic Cholera,' p. 444.

[2] The best known observations on this point are those that were made on the sudden outbreak of cholera, after heavy rains in April, at the celebrated Hurdwar shrine, situated at the confluence of the Jumna and Ganges. Brown ('Indian Annals of Med. Sc.') gives an account of an outbreak in Nepaul, in May, 1856, after heavy rains in April.

[3] This sudden extinction of the sickness, after the occurrence of very copious rainfall, happened in the Nepaul epidemic of 1856, mentioned in the foregoing note. It happened also, according to Puckle ('Brit. Med. Journ.,' July, 1876, p. 55), in the epidemic of 1874, at Bangalore, Mysore, and other high-lying places in the Deccan; the epidemic broke out in July, in severe heat and drought, and was extinguished in August under copious rains. "Similar phenomena," says Macnamara (l. c.), "are witnessed on a large scale in the province of Bengal."

[4] The examples of this are extremely numerous; thus, the epidemics of 1831 and 1832, in Germany, those of 1834 and 1835 in the South of France (Robert, 'Gaz. méd. de Paris,' 1835, No. 27), that of 1836 in Italy (Zarlunga, in Severino, 1836, Dec.), and that of 1849 in England. The observations made on this point during the North German epidemic of 1873 will be found in the history of the epidemic drawn up by me in the report of the Cholera Commission for the

that rule, too, has numerous exceptions. Sometimes the epidemic increases as the rain continues;[1] and still more frequently it first shows itself when rain follows upon a long period of drought.[2]

These apparent inconsistencies in the behaviour of the disease with reference to moisture may be reconciled by keeping the following facts in view. The influence of this causal element is an indirect one, or one that acts through the soil; a certain degree of moisture in the ground constitutes, as we have already indicated, an essential condition for the occurrence of cholera as an epidemic; absolute dryness as well as complete saturation of the ground will accordingly hinder the development of the epidemic or will bring it to an end; and therefore the question is one not merely of the amount of rainfall, but also of the physical character of the soil. The dry sandy soil of Madras and

German Empire (Berlin, 1879, VI,); my *resumé* of these observations is "that the development and increase of the epidemic kept pace with the progressive drying of the soil, the latter being ascertained either directly by measurement of the subsoil water or by watermarks, or from data as to the dryness of the summer, and the drying up of marshes, ponds, and wells."

[1] As at Quedlinburg in 1866 (Wolff, 'Geschichte der Cholera-Epidemie des Jahres 1866,' Quedlinburg, 1867, p. 13) and at Zürich in 1867 (Zehender, 'Bericht über die Cholera-Epidemie des Jahres 1867,' Zürich, 1870).

[2] Thus, Adams and Welch ('Army Med. Report for the Year 1864,' p. 346) state with reference to the Malta epidemic of 1865, that the disease began to be more generally diffused on the setting in of rain, and that it died out after a very large quantity of rain had fallen. Ferrini ('Saggio sul Clima e sulle precipue Malattie della Città di Tunisi,' Milan, 1860, p. 272) draws attention to the fact that in the Tunis epidemic of 1849-50 the deposition of moisture was always followed by an increase in the number of the sick. The outbreak of cholera in Luxemburg in 1865 was preceded by two years of almost absolute drought, so that much of the ground was as if scorched; the first considerable rains fell in October, and forthwith the disease, which had at first been observed only in isolated cases, became general. Further, the somewhat heavy falls of rain in March and July, 1866, were followed by a fresh accession of the epidemic. Cornish notices ('Med. Times and Gaz.,' 1868, March, p. 339) the occasional epidemic prevalence of cholera on the Malabar coast during the monsoon rains, but he states also that, under other circumstances, the heavy rains of the south-west monsoon may bring about the extinction of the sickness. He adds, in explanation, that "slight showers of rain at the end of the hot season may always, I think, be regarded as dangerous, as they afford the requisite amount of moisture to the surface soil (in combination with its existing high temperature) to favour the development of the germs of the disease."

the heavy clays and laterites of the North-West Provinces differ widely from the alluvium of Lower Bengal in their saturation point, and consequently in the opportunites that they afford for the development of an epidemic of cholera. But, finally, we must take into account the state of the *sub-soil water*, in so far as it is independent of the direct deposition of moisture, or when it is at a certain depth from the surface of the ground. It is obvious that when the sub-soil water stands at a low level, copious rains will produce an effect altogether different from that of moderate rains when the level of the subterranean water is high, and *vice versâ ;* and these proportionate results will be still further varied according as the soil is a highly porous one (as coarse-grained sand) or a moderately pervious one (such as loam or limestone marl).

This is the standpoint, in my opinion, from which we should consider the question of the influence of the wetness of the soil—whether that be due to rainfall and dewfall or to a rise of the sub-soil water not directly dependent thereon but dependent on other causes,—and of the quality of the soil, upon the epidemic diffusion of cholera. *It is always an essential circumstance that the soil be saturated, but only to that degree at which it is still pervious to air, and that the organic matters accumulated in it should undergo decomposition under the influence of somewhat high temperatures.*[1] Consequently the question is not as to the extent of the stratum of soil, saturated with moisture and permeable to air, or, in other words, a question of the higher or lower level of the sub-soil water ; but it is a question whether such a stratum exists at all, and that is, in fact, the gist of the much quoted and much misunderstood doctrine of Pettenkofer as to the *significance of the height and fluctuations of the sub-soil water for the production and diffusion of the acute infective diseases, cholera in particular.*

"In my view," says Pettenkofer in one of his later papers,[2] "the level of the sub-soil water reveals nothing more than this, viz. the limits of a certain degree of humidity in a porous stratum of soil, or the limits within which the pores are kept constantly full of water and all the

[1] Compare the observation of Cornish quoted above (p. 464, note).

[2] 'Zeitschrift für Biologie,' 1870, vi, p. 527.

air driven out of them. Between that degree of humidity and absolute dryness of the porous stratum, there are all those gradations when the pores are filled in part with air and in part with water in varying proportions, which we include all together under the terms "moist" or "wet." The point at which the pores are completely closed by water is one that may be observed with ease and certainty; and I have therefore chosen the level of the sub-soil water merely as an easily seen guage and index of certain states of humidity in the stratum of porous and permeable soil which overlies the sub-soil water, an index, namely, of the fluctuations in the state of humidity within a given period, and of the time that any one degree has lasted. Whether that index is a few feet nearer to or farther from the surface does not affect the value of its revelations. For the value of the index lies in this, that it declares the changes in the humidity of the overlying strata, by means of the natural effects of those changes. The fluctuations in the level of the sub-soil water have a meaning for etiology, only because they are traced back to those primary influences by which air and water are made to share, in varying proportion, the possession of the pores of an impregnated soil. Beyond that they have no significance. . . . Looked at by itself and for its own sake, the condition of the sub-soil water has as little significance as the hands and dial of a watch dissociated in thought from the works to which they belong."

In perfect agreement with this conception of Pettenkofer's, and with the view previously developed by me on the etiological import of the level and fluctuations of the sub-soil water for the production of cholera, are the conclusions that Lewis and Cunningham have arrived at after eight years (1870-77)[1] measuring of the sub-soil water in Calcutta. The highest level of the sub-soil water is reached in September, it falls steadily from that date to May, when it begins to rise gradually. Thus, the maximum prevalence of cholera corresponds with the lowest, and the minimum with the highest level of the sub-soil water. There is, however, no correspondence as between one year and another or one month and another; or, in other words, the absolute height of the sub-soil water is *by itself* of no significance for the amount of sickness. "If the concurrence of a low water-level," says those observers by way of summary, "and high prevalence of cholera in Calcutta be more than a mere coincidence—if any causal relation exist between the two phenomena—it cannot be a direct simple one dependent on the mere mass of water in the soil." They further call

[1] Op. cit., p. 28.

attention to the fact that the two years—1871 and 1872—in their eight years' period, which had the minimum number of cholera cases, were distinguished by the remarkably high level of the sub-soil water.

§ 113. Barometric Pressure.

Any causal relation between the degree of atmospheric pressure (height of the barometer) and the outbreak or spread of cholera, must be considered as out of the question, according to all our experiences hitherto. At all events, there is not the smallest correspondence to be made out between the level (or the fluctuations) of the atmospheric pressure and the course of an epidemic. Thus a very low barometer was noted in the epidemic of 1833 at Christiania,[1] of 1837 at Stralsund,[2] and of 1848 at St. Petersburg ;[3] during the prevalence of the disease at Aarau in 1854[4] there was an unusually high degree of atmospheric pressure; again, in the Königsberg epidemic of 1831, a decline in the number of cases always showed itself when there was a low pressure ;[5] during the epidemics of 1831 and 1837 at Darkehmen,[6] that of 1853 at Newcastle,[7] and that of 1854 at Oxford,[8] there was more or less of high pressure ; at the time of the cholera in London and Vienna in 1849, and at Munich in 1854, no variations from the normal barometric level could be ascertained. The observers in India have come to the same negative result ; for example, Messrs. Lewis and Cunningham conclude from nine years' observations: "there is no indication of the existence of any definite relation between degree of atmospheric pressure and prevalence of cholera."[9]

[1] Report in 'Eyr,' 1834, ix, p. 257.

[2] Anderssen, in 'Rust's Magazin,' liv, p. 499.

[3] Müller, 'Einige Bemerkungen über die Cholera,' Hannover, 1848.

[4] Zschokke, in the 'Schweiz. Zeitschr. f. Medicin,' 1854, pp. 371-72.

[5] Baer, in the 'Verhandl. der med.-phys. Gesellschaft über die Cholera in Königsberg,' i, No. 3.

[6] Carganico, in 'Rust's Magazin,' liv, p. 10.

[7] Hingeston, in the 'Assoc. Med. Journ.,' 1853, Oct., p. 927.

[8] Acland, 'Mem.,' p. 53.

[9] Op. cit., p. 16.

§ 114. Wind.

Concerning the *wind*, finally, as a factor in the production of cholera, we have to take into account its influence on the temperature and degree of moisture of the air, as we have already specially noted in speaking of the south-west monsoon as influencing the diffusion of cholera in Hindostan. We shall have to consider in the sequel whether the wind, as a carrier of the morbid poison, plays any part in the diffusion of cholera, and, if so, what.

§ 115. Mortality unaffected by Climate.

Just as cholera has come to be uniformly diffused over the globe, in all latitudes and under the most various conditions of climate, so also has it always and everywhere borne the stamp of perfect uniformity in its type and in its evolution. It has been the same disease on the marshy soil of the Gangetic valley, the saline steppes of Central Asia, the high table lands of Armenia and Algiers, the sandy plains of North Germany, the prairies of North America, in great epidemics and in small, and in sporadic cases as well as in epidemics; and thus it has shown itself, in respect of its type, to be unmodified by the latitude and longitude, and by the climate of the country, district, or place which it has invaded.

There are, doubtless, some not unimportant differences brought out on comparing the various cholera epidemics with respect to the range and severity of the disease, in its outbreak and its progress, at various places and at various times.[1]

[1] The whole of the statistical data made use of in the sequel are, I am convinced, wanting in trustworthiness, especially in so far as relates to the number of cases of sickness. But inasmuch as they are all alike subject to the same defect, they furnish at least the means of comparison with one another; and the best evidence that they possess that degree of usefulness is that their results come out very nearly the same, or at least vary within narrow limits.

In the first general outbreak in India the mortality among the English troops amounted to from 20 to 40 per cent. of the cases of sickness;[1] among the troops in the Bombay Presidency, again, 33 to 50 per cent. of the sick died in the years 1830-38, and in later epidemics, the mortality among them rose to 66·3 per cent. and upwards.[2] At Aden the proportion of the deaths from cholera was far greater in 1846 than in the epidemic of 1832,[3] and the same was observed in the Mauritius in 1854 as compared with 1819. At Königsberg, Magdeburg, and many other places in Germany, the disease was much more widely spread and more intense in 1848-49 than in 1831. In Lower Austria the cholera of 1836 was more widely spread than in 1831 and 1832, but it was milder in character.[4] In the department of the Meurthe the cholera was more general in 1832[5] than in 1849, but in the latter year it was more virulent. In Glasgow the mortality in 1832 was 41 per cent. but in 1849 only 22 per cent. In New York the type of the disease was much milder in 1834 than in 1832.[6]

Those differences in the amount of sickness and in the type of the disease in the several epidemics are explained partly by the influence of the weather, and partly by other factors which from time to time increase or diminish the predisposition of the population to the malady. But in the majority of instances we seek in vain for an explanation of the fact, such as can be rationally substantiated by evidence; we are face to face with the old pathological riddle that has confronted us in the epidemics of smallpox, scarlet fever, and measles, which are at one time slight and at another time severe. We may indeed assume a greater or less degree of concentration of the morbid poison, and therein find a solution of the problem, which is, of course, not a proof, but a mere hypothesis. At all events, it is just as impossible to explain these differences for cholera, as for those other diseases, by means of climatic influences and geographical

[1] Kennedy, 'Notes on the Epidemic Cholera,' Calcutta, 1827, p. 79. In several reports the still more favorable estimate of 10 and even 5 per cent. is given; but, as Kennedy explains, these data are not to be trusted. "When the disease first appeared," he says, "there were many causes tending to magnify the number of attacks and the number of cures, and a most erroneous estimate was too generally formed of the relations in which these events actually stood to each other."

[2] Hunter, in the 'Lond. Med. Gaz.,' 1846, Oct., p. 595, 1847, Jan., p. 11.

[3] Ib., 1846, July, p. 82.

[4] Report in the 'Oest. med. Jahrb.,' n. s., xix, p. 74.

[5] Simonin, 'Recherch. topogr. et méd. sur Nancy,' p. 122.

[6] Lee, in the 'Amer. Journ. of Med. Sc.,' 1834, Nov., p. 256.

position; for the variations occur equally in all latitudes. It remains true, however, as the following table shows, that the mortality from cholera comes, on the whole, to very nearly the same figure at all places where the disease has spread to, amounting, in the mean, to 50—60 per cent.

Percentage of Deaths from Cholera in Various Countries.

Place.	Time.	Deaths in 100 of the sick.
India } Presidency of Bengal[1]	1860—1873	64·85
India } ,, ,, Bombay	,,	59·2
India } ,, ,, Madras	,,	53·41
Malta	1865	60·36
Italy	,,	54·70
Roumania	1865—1866	47·45
Pesth	1872—1873	47·15
Belgium	1866	52·16
Prussia	1831—1832	57·43
,,	1852	58·94
,,	1866—1867	51·77
,,	1871	55·18
,,	1873	51·97
Russia (European)	1854	48·98
Sweden	1855	57·52

§ 116. Predisposition of Individuals, Races, and Nationalities.

Let us now consider in a few words the question of *individual predisposition* as an influence in the spread of cholera. It is difficult to form a reliable judgment as to the significance which attaches in this respect to peculiarities of *race* and *nationality*, inasmuch as the elements entering into a mixed population are very variously circumstanced in matters of food, habitations, and living in general. It always resolves itself, therefore, into a question how far the amount of sickness and mortality from cholera among the various constituents of the population is an affair of nationality or of social well-being; and it is all the more

[1] These data from India relate exclusively to the British troops (Native and European).

difficult to decide the question because of the numerous contradictions that we encounter in the statements bearing upon it. There is an almost complete unanimity that the *negro race* incurs the greatest danger from cholera; Christie[1] expresses himself to this effect after an experience gained on the East Coast of Africa, Dauban from his observations in the Mauritius, and Petit[2] and Vinson[3] with reference to disease among the negroes in Réunion.

On the East Coast of Africa the mortality amounted to 6·5 per cent. of the population among the Hindoo coolies, 10 per cent. among the Arabs, but 25 per cent. among the negroes. In Mauritius, according to Bonsergent,[4] 22,000 negroes died within two months in the epidemic of 1819; in like manner, in the epidemic of 1854, the African race furnished by far the largest contingent to the sick-list and death-roll, while there were only isolated cases of death among the Chinese, notwithstanding that they lived in the district most severely affected.[5] The report of Petit on the sickness among the negroes in the Réunion epidemic of 1859 is: "They fell down in a stupor as if they had been paralysed . . . men who had been full of life and vigour immediately before, were prostrated in the course of three or four hours' time."

In the Dutch East Indies the mortality from cholera in 1873 amounted among the natives to 35·3 per cent. of the sick, 51·5 per cent. among the Europeans, while it was nearly 60 per cent. among the negroes. In Ceylon, during the years 1859-67, an average of 4·9 per 1000 sickened among the European troops, and 2·38 died of the disease, while the numbers among the negro troops were 8·7 and 4·35 respectively.[6] According to the statements of Walther,[7] there died in the 1865 epidemic in Guadeloupe 2·7 of the Chinese population, 3·26 of the Hindoo coolies, 4·31 of the whites, 6·32 of the mulattoes, and 9·44 of the negroes. In like manner, in the epidemic of 1863 in Brazil, it was the negro race, as we learn from Aschenfeld,[8] that ran the greatest risk. In the United States Army the mortality in the epidemic of 1866 was 77 per thousand among the white troops and 135 among the black.

On the other hand, there is less agreement among Anglo-Indian physicians as to the relative immunity from cholera which some observers allege that the Hindoo enjoys as com-

[1] 'Lancet,' 1871, Feb., p. 188.

[2] 'Revue maritime et coloniale,' 1861.

[3] 'Bull. de la Soc. des Scienc. et des Arts de la Réunion,' 1871.

[4] 'Observat. . . . . sur les maladies . . . chez les noirs a l'île Maurice,' Montpellier, 1837.

[5] Clerihew, in the 'Med. Times,' 1856, Aug. 9th.

[6] I have taken these details from the 'Army Medical Reports.'

[7] 'Arch. de méd. nav.,' 1869, Aug., p. 104.

[8] 'Virchow's Archiv.,' vol. 28, p. 414.

pared with the European. From the reports of Cunningham, Bryden and others, sickness from cholera appears, at all events, to be more frequent among European troops stationed in India than among the sepoys; but it is very questionable whether the reason of that is to be really looked for in national peculiarities, and not rather in the different mode of life of the two classes of troops—simpler and more purpose-like among the natives than it is among the English. This much, at least, is certain, that the type of the disease is a tolerably uniform one, whether among the white troops or the native. The following are the proportions of sickness and mortality, compiled from several years' observations:

	European Troops.			Native Troops.		
	Sickness per 1000 men.	Mortality per 1000 men.	Mortality per 100 of the sick.	Sickness per 1000 men.	Mortality per 1000 men.	Mortality per 100 of the sick.
Presidency of Bengal	28·7	9·7	33·70	5·3	1·6	30·19
„ „ Bombay	26·4	8·6	32·53	9·6	3·2	33·33
„ „ Madras	19·8	6·9	34·85	13·5	5·8	42·96
Mean for whole country	24·96	8·40	33·69	9·46	3·53	35·50

According to this, the mortality relative to the sickness is actually greater among the sepoys than among the European troops; the death percentage given by Chipperfield[1] for the Madras epidemic of 1866 bears this out, the deaths among the Europeans being 42·8 per cent., and among the Hindoos 49·24. All observers agree that in Lower India (Anam, Siam, and Burmah), the native population, with their poor means of livelihood, suffer much more from cholera than the European colony.[2]

In this question of individual predisposition to cholera-sickness, the stress must always be laid on the amount of resistance-power in the organism; experience has shown that those groups or classes of the population, most exposed to the injurious influences that appear to be especially adapted

[1] 'Madras Quart. Journ. of Med. Sc.,' 1867, xi, p. 37.

[2] Cf. Thil, 'Remarques,' p. 29; Richaud, in the 'Arch. de méd. nav.,' 1864, May, p. 340; Houillet, ib., 1867, Dec., p. 405; Gimelle, in the 'Union méd.,' 1869, i, p. 569.

to weaken or destroy the power of resistance, are at the same time in greatest risk from the disease. The bearing of this etiological factor on the production of the morbid condition is most strikingly brought out in the prevalence of cholera among the *proletariat*, which has always and everywhere supplied by far the largest contingent of victims; it has not unfrequently been almost the only part of the population to suffer, and, as Melzer expresses it,[1] it has been "the true quarter-master of cholera."

§ 117. Nature of the Morbid Poison, Mode of Reproduction, and Manner of Diffusion.

In tracing the history of cholera, I have started from the assumption, which hardly anyone nowadays would call in question, that the disease is indigenous only in certain regions of India, and that its diffusion beyond those foci has always been associated with the importation of the poison that underlies the disease. This poison, however, is capable of reproducing itself beyond its native habitat, and the steadily increasing area of the disease has depended upon the continual introduction of the poison from country to country and from continent to continent. This potency of the cholera poison, and its power of reproducing itself, are bound up, as the foregoing inquiry shows, with certain exterior conditions; and it is variations in these that serve to explain the intermissions in the continuity of the disease or its remissions and exacerbations, or, in other words, the appearance and disappearance of the sickness from time to time as an epidemic. But while this reproductive power of the poison is kept up continuously in those regions where it is indigenous, it is sooner or later exhausted outside the endemic foci; and a fresh outbreak of the disease, after it had been completely extinguished, always presupposes a new importation of the poison from its native habitat. These assumptions and conclusions are strictly based upon the facts which we have unfolded in the preceding pages. But now arises a series of questions which are directed to the nature of the (supposed) morbid poison, to the manner of its repro-

[1] 'Prager Viertelj. für Heilk.,' 1851, iii, p. 132.

duction, and to the media by which it is carried; and to answer those questions, our experience hitherto has hardly afforded us adequate materials.

Now that the infectious character of cholera, and the communicability and reproduction of the morbid poison, have been accepted as the general belief, there can be no longer any doubt that this morbid poison must be an organic one, or, in other words, an organised body, and that the disease must be reckoned among the zymotic or *parasitic* processes. This opinion gained a certain currency when the disease first broke out on European soil; in fact, there were those who did not hesitate to set down "the peculiarly-tinted, mist-like obscurities in the air," which were observed at several places to precede an outbreak of the disease, as dense swarms of lower organisms—"cholera-animalcules"—that had been carried from India by the wind and were the means of spreading the disease as a pandemic. The conjecture supported itself upon Ehrenberg's observations on the wide dispersion of infusoria by the wind. But this charmingly simple view of the matter was given up after a time, especially when Ehrenberg himself examined with the microscope the particles in the air of cholera-stricken houses, and declared the hypothesis to be a piece of silliness. Others, again, such as Thomson,[1] arrived at a negative conclusion after very thorough chemical and microscopical investigation of the matters present in the air of cholera wards. Still, the theory of the parasitic nature of the disease seemed to be one that was entitled to all respect; and the investigation, changing its direction, has taken up the more likely and more rational business of searching the sick organism itself, especially the dejecta, the blood, the intestine, &c., for lower organisms, such as might be brought into causal connexion with the diseased process.

Böhm[2] was the first to institute inquiries of that kind, and to call attention to the presence of cryptogamic bodies, resembling the ferment-

[1] His first investigations were made in the epidemic of 1849, and later ones in 1854; the results are published in the 'Report of the General Board of Health on the Epidemic Cholera of 1848-49,' London, 1850, p. 119 (also in 'Med.-Chir. Trans.,' 1850, xxxiii, p. 67), and in the 'Lancet,' 1856, Jan., p. 63.

[2] 'Die Kranke Darmschleimhaut in der asiatischen Cholera,' Berlin, 1838, p. 57.

fungi, among the dejecta and in the intestine. Brittan[1] believed that he had arrived at a more definite result when, working in conjunction with Swayne in the Bristol epidemic of 1849, he met with small "annular" bodies in the dejecta of cholera patients; these he always missed in the dejecta of healthy persons or of those suffering from other diseases, but he found them, as he thought, in the air of cholera wards. Bennett[2] confirmed these statements, in part at least, in his investigations at Edinburgh the same year. About the same time, Pouchet[3] also had found lower organisms in the excreta of cholera patients, which he described as vibrios belonging to the species *Vibrio rugula*; Davaine, again, designated the living organisms found by him in the dejecta of cholera as belonging to the genus "Cercomonas;" while Pacini[4] described, as a discovery of his own made in 1854 and 1856, small granular bodies composed of round cells and resembling the *Bacterium termo* of Dujardin. Then followed the observations of Ercolani,[5] who did not profess to decide whether the vegetable formations that he had found in the excreta were the actual morbid poison or merely the carriers of the same. Next came the investigation of McCarthy and Dove,[6] who found in the fresh dejecta large masses of cellular elements, embedded in flakes of mucus and endowed with active powers of movement. Much interest was excited by the result of the inquiries which three German investigators, Klob,[7] Thomé,[8] and Hallier,[9] had instituted in the epidemic of 1866 on the formed elements contained in cholera stools; besides various cells of fungi, the *Bacterium termo* demonstrated by Pacini (the *Zooglæa termo* of Cohn) was found to be present in large numbers. Hallier in particular believed that his micrococcus-doctrine was the final solution of the question concerning the parasitic nature of the cholera poison. However, further inquiries in this field, and the criticism of competent judges, soon proved how little any definite conclusion as to the nature of the cholera poison was justified by the facts ascertained up to that time. The doctrine of Hallier received a complete refutation at the hands of De Bary[10] and Fr. Cohn.[11] Virchow published a case of arsenical poisoning in which the microscopic examination of the contents of the intestine showed "innumerable masses of the most minute bacteridia and vibrios corresponding in every particular to the cholera-

[1] 'Lond. Med. Gaz.,' 1849, Sept.

[2] 'Monthly Journ. of Med. Sc.,' 1849, Nov., p. 1235.

[3] 'Gaz. méd. de Paris,' 1849, p. 327.

[4] 'Observaz. mikrosk. e Deduz. pathol. sul Cholera Asiatica,' Firenze, 1854; and 'Della Natura del Cholera Asiatica,' Firenze, 1866.

[5] 'Annali univ. di Med.,' 1866, vol. 195, p. 375.

[6] 'Lond. Hosp. Reports,' 1866, iii, p. 445.

[7] 'Pathol.-anat. Studien über das Wesen des Cholera-Processes, Leipzig, 1867.

[8] In 'Virchow's Archiv,' 1867, vol. 38, p. 221.

[9] 'Das Cholera-Contagium,' Leipzig, 1867; see also 'Centralbl. f. d. med. Wissensch.,' 1867, No. 30.

[10] In the 'Jahresbericht' of Virchow and Hirsch, 1867, ii, pp. 240—252.

[11] 'Bericht der Schlesischen Gesellschaft,' 1868, pp. 19 and 52.

fungi described by Klob and others."[1] In perfectly fresh cholera dejections, De Bary had met with bacteria and allied forms only in very small numbers, while they had multiplied enormously after the material had stood for a day or two in closed vessels; and, on the other hand, he had found the same objects moderately abundant in the dejecta of a patient suffering from simple diarrhœa. A like negative result was arrived at by Kyber.[2] Nor have the investigations instituted by Lewis and Cunningham in India led to a definite conclusion on the question; the bacteria and vibrios (including Hallier's micrococci) that were found in larger or smaller quantities in the excreta of cholera patients differed in no respect from those occurring in other putrefied fluids; and, in the cultivation-experiments made with them, not only did the specific "cholera-fungus" of Hallier remain altogether absent, but only those forms of fungi were found, which developed on putrefying organic substances at large, in the same place and at the same time. Moreover, controlling observations with the intestinal discharges of individuals not suffering from cholera revealed the presence of the same objects as in the cholera dejecta. Indeed, bacteria and the leptothrix chains of Hallier often occurred in them in even larger quantities than in the dejections of cholera; while the cultivation-experiments gave the same result in the one case as in the other.

But although the inquiries designed to prove the parasitic nature of the cholera poison have been hitherto unfruitful in results, we ought by no means to consider the question as settled in the negative sense. We cannot but admit that the theory of the production of the disease, founded upon that assumption, adapts itself to the facts with less constraint than any other, that no fact in the history of cholera goes against it *absolutely*, and that it finds support in many remarkable analogies. It ought therefore to be maintained, and made the subject of continued exact inquiry, until such time as it is either established by proof or shown to be absolutely untenable.

§ 118. Experiments to Communicate Cholera to Animals. Criticism of the Same.

A second series of experimental inquiries upon the cholera poison took the direction of showing that it *resided in the blood and in the secretions and excretions* of cholera patients, especially in the *dejecta;* and of proving the com-

[1] 'Virchow's Archiv,' 1869, vol. 47, p. 524.

[2] In the 'Dorpater med. Zeitschrift,' 1873, iii, p. 74.

municability of the disease by the device of *infecting animals experimentally*.

When the disease first appeared on European soil, the view gained currency that the poison, having penetrated into the organism, multiplied therein in the course of the disease-process, and was eliminated with the dejecta of the sick; and, consequently, that the spread of the disease was closely connected with the implied conveyance of the poison from person to person.[1]

Thus Hildebrand,[2] among others, published a paper in which he said that "dogs, cats, and poultry which had consumed the dejecta of cholera patients had died under symptoms like those of cholera, while others had swallowed the same material without any ill effect;" and he adds that "these observations, whether obtained by pure chance, *or from experiments purposely made* (some physicians flattering themselves that they could thereby settle the dispute about contagiousness), leave a very conflicting impression." Experiments to infect animals by inoculating with the blood of cholera patients were first made by Namias,[3] who thought that he had succeeded; while Novati[4] and Semmola,[5] experimenting in the same way, came to a negative conlusion. Magendie,[6] also, who had injected the blood of a cholera patient into the veins of a dog, declared that the animal sickened with symptoms like those of cholera. That kind of infection-experiment began to be made on a larger scale about 1854. The form of the experiment was mostly shaped on the assumption that the morbid poison resided essentially in the dejecta of the sick; the dejecta were introduced into the experiment-animals (monkeys, pigs, dogs, rabbits, mice, pigeons, and even frogs) partly with their food, partly in enemata, or by injections into the trachea, or by subcutaneous injections. The experiments made upon white mice by Thiersch,[7] with minimal doses of the dejecta, excited a good deal of interest; they were received as highly satisfactory, and they at the same time gave colour to the belief that the discharges of cholera patients were more certain to induce choleraic sickness and the death of the animal, if they had been

[1] Pellarin has repeatedly claimed for himself the priority of the doctrine that the disease is communicated through dejecta containing the morbid poison. This contention, however, is based, as we shall see, upon an error.

[2] 'Oest. med. Jahrb.,' 1838-39, n. s., xvii, p. 446.

[3] 'Annali univ. di Med.,' 1836, lxxix, p. 162.

[4] 'Giornale per servire ai progr. della Pathol.,' 1836, v, p. 373, vi, p. 207.

[5] 'Annali univ. di Med.,' 1838, lxxxiv, p. 150.

[6] 'Vorlesungen über epid. Cholera.' From the French. Berlin, 1839, p. 80.

[7] 'Infections-versuche an Thieren mit dem Inhalte des Cholera-darms,' Munich, 1856. In abstract in the 'Hauptbericht über die Cholera-Epidemie des Jahres 1854 in Bayern,' Munich, 1857, p. 560.

permitted to undergo a moderate degree of decomposition, beyond which they would be harmless. Simultaneously, Lindsay[1] made experiments on dogs, in some of which he injected the blood of cholera patients under the skin, and in others introduced large quantities of the dejecta into the body. The consequences were negative, but, on the other hand, symptoms like those of cholera appeared in some of his animals when he exposed them to exhalations from the stools or vomit, and from linen that had been saturated with the perspiration of the sick. Burdon Sanderson[2] repeated the experiments of Thiersch, and was able to confirm his results; also Crocq,[3] Legros and Guyon,[4] Leyden,[5] and more lately Popoff,[6] have experimented with success.

But there have to be set against these positive achievements, a much larger number of experimental researches that have failed. Schmidt,[7] in 1850, practised injections into the veins with the blood of cholera patients, and introduced the discharges into the animal's body, but he arrived at a conclusion in the absolute negative. Meyer[8] was a little more fortunate, but the result of his experimentation was after all only a doubtful one. Quite without result, again, were the researches of Guttmann and Baginski,[9] of Bartolow,[10] of Woodman and Heckford,[11] of Stockvis,[12] Snellen and Müller,[13] of Patterson,[14] of Lewis,[15] and of Ranke;[16] the latter experimented exactly after the method of Thiersch, and as he obtained a negative result he was able to repel Thiersch's charge[17] against Stockvis, the latter having stigmatised the experiments as fallacious although he had not followed his (Thiersch's) method. We find a true reflex of these contradictory results of the experiments on infection in the paper by Högges,[18] who experimented on dogs, rabbits, and guinea-pigs, and who obtained, under the most various ways of introducing the substance, appearances of an inflammatory disorder of the mucous membrane of the stomach and intestines, a certain number of the experi-

[1] 'Edin. Med. and Surg. Journ.,' 1854, April, p. 275, Oct., p. 630.
[2] 'Lancet,' 1867, Oct., p. 488, and p. 530.
[3] 'Bull. de l'Acad. de Méd. de Belgique,' 1866, p. 916.
[4] 'Journ. de l'Anat. et de la Physiol.,' 1866, p. 584.
[5] In Wiewiorowsky, 'Disser. de Cholera Asiatica, &c.,' Königsberg, 1866.
[6] 'Berl. klin. Wochenschr.,' 1872, No. 33.
[7] 'Characteristik der epid. Cholera,' Leipzig, 1850, p. 79.
[8] 'Virchow's Archiv.,' 1852, iv, p. 29.
[9] 'Centralbl. für d. med. Wiss.,' 1866, No. 44, and 'Berl. klin. Woch.,' 1867, No. 8.
[10] 'Cincinnati Lancet and Obs.,' 1866, ix, p. 652.
[11] 'London Hosp. Rep.,' 1866, iii, p. 477.
[12] 'Tijdschr. voor Geneesk,' 1866, ii, p. 284.
[13] 'Nederl. Arch. voor Natuur-en Geneesk,' 1867, iii, p. 51.
[14] 'Med. Times and Gaz.,' 1872, April, p. 472.
[15] 'Indian Annals of Med. Sc.,' 1872, Jan., p. 443.
[16] 'Bayr. med. Intelligenzbl.,' 1874, No. 15.
[17] 'Zeitschr. für Biologie,' 1867, iii, p. 137.
[18] 'Centralbl. f. d. med. Wiss.,' 1873, No. 50, 51.

ment-animals dying of it. I am unable, for my own part, to infer from these data that we have here really to do with a choleraic disease in animals. How little satisfied M. Högges himself was with the results that he obtained, appears from his own words:[1] "After having endeavoured to get some closer insight into the action of choleraic discharges by the aid of experiments designed from various points of view, I must confess that the question seems to me not less confused than it did before the experiments were begun."

It is perfectly clear that nothing has been gained by these experiments towards deciding the question whether cholera is communicable, and still less for deciding the question whether the cholera poison resides in the dejecta of the sick. The experiment-animals were subjected to unusual conditions of living; they were not rarely exposed to tortures; blood-serum and bowel-discharges were poured through them by the ounce. It is not so remarkable that a few of them sickened, as that the greater part of them did not perish. The sick animals showed signs of an affection of the stomach and intestine; but neither from the aggregate symptoms nor from the anatomical condition does it follow with certainty that this affection actually corresponded to the process of cholera in man. On the other hand the negative results, in my opinion, are not decisive for the solution of the problem proposed, inasmuch as it is still unsettled whether those classes of the animal kingdom from which the experiment-animals were selected, possess any susceptibility to the cholera poison at all. On that point evidence is entirely wanting; and the want of it can hardly be made up for by the observations that have been published upon (assumed) choleraic sickness among the domestic animals, poultry, &c., at the time of an epidemic of cholera, inasmuch as these are completely unreliable. Moreover, in so far as the researches seek to prove the general communicability of cholera, they have lost all importance; for epidemiological observations leave no manner of doubt on the matter. A special value attaches to them only in so far as they are calculated to furnish evidence on particular points. These questions are: whether the secretions and excretions of the sick person, and especially the alvine discharges, are the vehicle of the morbid poison; whether the poison repro-

[1] 'Allg. Zeitschr. f. Epidemiol.,' 1874, Pt. 2, p. 81.

duces itself within the organism of the sick; or whether the individual (sick or healthy) is a carrier of the cholera poison only in so far as it clings to him externally, the reproduction taking place outside; or, in the words lately introduced by Pettenkofer, whether cholera belongs to the endogenous or the ectogenous processes of communicable disease.

We have become so used to the notion of the cholera poison multiplying within the bodies of the sick, and of the choleraic discharges in particular being the carriers of the same, that we hardly appreciate nowadays how slight a foundation there is for that opinion. A certain number of facts have been brought together from practical experience in support of it: such as the concentration of sickness in places where cholera dejecta had been deposited; cases of illness among persons who had been employed in emptying cesspools which had received the evacuations of cholera patients; house epidemics limited to such of the inhabitants of a quarter as had used one privy, or whose privies opened into one cess-pit; progress of the epidemic along streets or quarters of the town which had one common system of canalisation. But, of all such facts, the greatest importance attaches, in my opinion, to an observation that has been often made and verified, viz. the outbreak of cholera in a locality spared hitherto, and in fact situated at a distance from the centre of the pestilence, when linen, bedding, or clothes, soiled by the dejecta of cholera patients, had been brought to it, the disease attacking just those very persons who had come into direct contact with the tainted articles. At the same time, I should not by any means wish to ignore the view that even such facts may be otherwise interpreted in favour of the ectogenous production of the disease.

§ 119. The Question of Contagiousness.

However probable, then, the infective property of the cholera-dejecta may be, it is not less certain that their actual disease-producing potency is dependent on certain exterior conditions, on some change that they undergo after their

elimination from the organism, or on the access of some factor extrinsic to them. We should not be justified, therefore, in reckoning cholera among the *contagious* diseases properly so-called, that is to say, among those diseases in which the morbid poison, after being eliminated from the sick and conveyed to other individuals, is able forthwith to unfold its infective power. We cannot but admit, as I have already stated elsewhere,[1] that even the contagious diseases, properly so-called, are to some extent dependent for their epidemic development upon exterior influences; smallpox and measles occur as epidemics, both in temperate and in tropical latitudes, more often and more extensively in the cold season than in the hot; smallpox, as well as typhus, takes up its abode most readily in those places where the noxious influences due to neglected hygiene make themselves most felt. But the relation of those factors of disease to highly contagious maladies of that sort is clearly different in kind from the influence of atmosphere and locality upon the diffusion of cholera. In the one case we appear to have to do with a heightening of the potency of the morbid poison by its concentration, or with a multiplication of the points of contact between the poison and the individuals susceptible to it, and possibly also with an exaltation of the individual predisposition. In the other case—that of cholera—the exterior influences are a *conditio sine quâ non* for the development of the disease into an epidemic. A person sick of typhus, smallpox, or measles may become under all circumstances a centre whence disease may radiate; but a cholera patient, or any other object charged with the cholera poison, will not become a centre of disease unless the above-named exterior conditions are present at the same time. A comparison of the diseases in question, as epidemics on a large scale, would bring out the essential differences between the mode of spreading of smallpox, measles, and typhus, on the one hand, and of cholera, on the other. But those differences stand out still more prominently in smaller and more easily-surveyed areas of observation, as, for example, in general hospitals. It is well known how many victims typhus has

[1] 'Berichte der Cholera-Commission für das deutsche Reich.,' Part vi, Berlin, 1879, p. 310.

often exacted among the physicians and nurses in hospitals for typhus, and it is also well known that there is much danger to the inmates of a hospital, or division of a hospital, into which typhus patients have been admitted. But the behaviour of cholera affords an almost complete contrast. Here it is the exception for the physicians, nurses, and servants in cholera hospitals to contract the disease; or, at all events, cases of sickness are not more common among them than among the inhabitants in general of a locality subjected to the epidemic. And it is precisely this fact that has been used to support the opinion which denies the communicability of cholera altogether, and includes it among the so-called "miasmatic" diseases.

"The prevailing facts and arguments," says Lyons, in a report on the cholera epidemic of 1872 at Chittagong,[1] "render the conclusion absolute that cholera is contagious; the completeness and consistency of the evidence will satisfy even the judicial mind. There was, however, a remarkable non-correspondence as regards the quality of contagiousness or communicability displayed by the disease outside and inside the hospital to which cases of cholera were removed directly they were discovered. I am unable to point out any instance that came under my personal observation in which the sick communicated the disease to their attendants or other persons in their neighbourhood."

§ 120. How far Dependent on the Soil—Epidemics on Board Ship.

The theory of the communicability of cholera by way of direct contagion is wanting in a groundwork of precisely ascertained facts. But, inasmuch as there can be no doubt of communicability itself, we must either assume that the cholera patient eliminates an infective substance, which, in its primary form, has no potency as a cholera poison, but only attains its specific infective power after it has undergone some kind of change or maturation outside the body

[1] 'Indian Annals of Med. Sc.,' 1873, Jan., p. 425.

under the influence of certain exterior agents; or, there is the alternative view that the multiplication (reproduction) of the cholera poison proceeds quite independently of the cholera patient (as such), clinging to persons (healthy or sick) or to other objects, getting carried by these from place to place, and giving rise to an epidemic outbreak wherever it meets with conditions suitable for its reproduction. Both hypotheses, in my opinion, are equally tenable *à priori;* but, in so far as I am able to form a judgment on the facts, the first ought to have the preference. The sort of conditions necessary for the ripening of the cholera poison have been already adverted to by me in speaking of the influence of atmospheric and terrestrial states upon the spread of the disease. But many points relating thereto are still obscure; even that question cannot be regarded as settled altogether finally.

A relatively high temperature is unquestionably a favouring influence in the production of cholera; but we must admit some very considerable, if not very frequent, exceptions to that general rule (*vide antea*, p. 460-62). The opinion which has several times met with some favour, that exceptions of that kind are to be explained by the artificial heating of houses in the winter time, appears to me to go against the simplest rules of logic; for, if that be the reason, we cannot help asking why the disease has survived the winter in a few rare cases only, while the artificial warming of houses has gone on every winter.

But we must hesitate just as much, in our capacity of impartial critics, to admit the *absolute* dependence of the pathogenesis upon the above-mentioned conditions of soil. Here again we are entitled to say that cholera flourishes *principally* on a soil that is porous and rich in organic detritus, after a partial drought, and under the influence of a relatively high temperature; and to say that the soil is so much the less favorable to cholera-production the farther removed it is in physical characters from this ideal of a cholera soil, or, in other words, the less the amount of organic matters accumulated in it, and the less favoured their decomposition by concurrent circumstances. But, on the other hand, we find a series of facts which prove that cholera may attain a wide diffusion

under other conditions. The mere fact of the disease breaking out as an epidemic in a particular group of houses, or even in a solitary house, or in a single flat, while the whole neighbourhood keeps quite free,[1] justifies a doubt whether it is the soil underlying the building that has constituted the breeding-place of the morbific agent, or whether it may not have been rather the walls, ceilings, and floors of the rooms in question that have taken the place of the soil, under certain circumstances which we cannot define particularly. May not these have served to take up organic matters, admitting them in a finely divided state into pores and holes and crevices, where they could undergo the same processes of decomposition as in the soil, under conditions otherwise similar, and where they would play the same part in the production of the disease?

The epidemic prevalence of cholera on board ship, which the partisans of the soil-theory altogether and unfairly ignore, affords evidence on this point which seems to me to be unanswerable. That cholera very seldom spreads to an epidemic, or has a long career as such on boards vessel on the high seas, is a well-established fact; it passes, indeed, as an axiom, based on many trustworthy observations, that there is no more certain means of combating the disease, when it has appeared among the crew or passengers of a ship lying in an affected port, than putting out to sea. In the epidemics of 1837 and 1849 at Malta, as well as subsequently in 1854-55 during the Crimean war,[2] this practice was adopted with the English war-ships with the best results. The same was proved by experience on a large scale in Indian ports (Calcutta, Bombay, &c.); the very large body of observations which comes to us from these waters go to

[1] I must expressly state that I do not refer to those instances in which we have to deal with explosive outbreaks of cholera, outbreaks in which the whole of the cases have followed close upon one another within a short time, where accordingly one must imagine a simultaneous infection due to the introduction of a morbid poison already grown ripe and potent; but I refer to those outbreaks which are protracted over a comparatively long period, and in which the several cases of disease, occurring at intervals the one after the other, stand in an obvious genetic relation.

[2] See Burnett, in the 'Med. Times and Gaz.,' 1855, Jan., p. 53, and Babington, 'Assoc. Med. Journ.,' 1856, Aug., p. 748.

prove how seldom cholera occurs at all on board ship,[1] and that in many cases, where the disease continued for some time after the ship put to sea, there was reason to suppose that the whole of the subsequent outbreaks of sickness were referable to a previous infection acquired while the vessel was in port. The immunity obtained by following that rule is in part due to the removal of the crew beyond reach of communication with the infected port; but it also depends without doubt upon the fact that the ship affords a soil little suited to the further development of the disease. More recent experiences, however, have furnished proof that this immunity is not an absolute one, that true epidemics of cholera on board ships do in fact develop from time to time. In such cases it stands to reason that an importation of the cholera poison from an infected shore by individuals or effects must be always presupposed; but the course of these epidemic points to a prolonged infection by means of successive reproductions and maturation of the morbid poison.

Passing over numerous communications bearing on this subject, in which the course of the disease is not indicated in a reliable way, I shall confine myself to giving a brief account of a few of the more instructive epidemics of cholera on board ship.

On the 3rd of October, 1853, a French three-masted ship sailed from Havre for New York with 560 passengers and 25 of a crew; some of the passengers had come to Havre from Rotterdam, where the cholera was then raging. On the 11th of October, one of the children sickened and soon died, on the 12th a sailor fell ill and died, then followed fifteen deaths from the 12th to the 22nd October, and six deaths from the 23rd October to the 1st November; so that, out of nearly 580 persons, twenty-two died of the disease, and at least 10 per cent. sickened. The outbreak lasted three weeks; and, among those that sickened or died, there were not only such as had come from Rotterdam to Havre, but also other passengers, and, as we have seen, at least one member of the crew.[2]

On board the steamship *Leibnitz*, which sailed from Hamburg for New York on the 12th of November, 1867, cases of sickness and death from cholera occurred first among emigrants from Mecklenburg, who had most probably introduced the disease into the vessel; but numerous

[1] See Pettenkofer, in the 'Zeitschrift für Biologie,' 1872, viii, p. 8; Waring, in the 'Med. Times and Gaz.,' 1867, May, p. 480; Macpherson, 'Cholera in its Home,' p. 42.

[2] Rösch, 'Württemb. med. Correspondenzblatt,' No. 5, p. 37.

cases of illness and death occurred afterwards among the other passengers, so that up to the end of December, or within a space of six weeks, 145 cases, with 105 deaths, had been observed, including only one of the crew.

On the 28th of March, 1866, the steamship *England* left Liverpool for New York with 37 cabin passengers, 1059 'tween-deck passengers, and 80 to 100 of a crew, in all some 1200 souls. Part of the 'tween-deck passengers had come to Liverpool from Rotterdam, and these were probably the source of the disease. The first cases occurred on the 2nd of April, and by the 9th of that month, when the vessel had reached the latitude of Halifax, 150 persons had been put on the sick list, of whom 46 had died. At Halifax, the sick from the *England* were transferred to a hospital ship, and the healthy put into quarantine; and among the latter, during the next two days (10th and 11th April) 100 new cases of sickness occurred with 40 deaths. It was not until the 30th of April that the disease disappeared completely. Of the 1059 steerage passengers, 500 to 600 in all must have been seized, and of these 280 to 300 died; the cabin passengers escaped altogether, and among the crew six deaths occurred. As the ship bore up for Halifax, she was hailed by a pilot who was cruising in a boat with two other men; he agreed to bring the ship into the quarantine harbour at Halifax, and, having learned that there was a fatal form of disease on board, he laid his boat close alongside, sent up his papers to the captain in a basket that had been lowered from the ship, and was then taken in tow. Having brought the ship up to the quarantine harbour, without having boarded her, he rowed ashore with his two comrades. On the night of the 10th-11th April, or two days after he had come thus remotely into contact with the *England*, he was taken ill of cholera, and three days afterwards cholera broke out in his family. Almost at the same time, one of his two companions sickened, and gave the disease to his three children. It stands to reason that this must have been an infection proceeding from the ship, inasmuch as at that time (April, 1866) the whole Western Hemisphere was, and had been for years,[1] quite free from cholera.

On the 13th of October, 1855, the emigrant ship *Franzisca* sailed from Hamburg for Rio Janeiro with 220 'tween-deck passengers, 13 officers and cabin passengers, and 16 of a crew. On the 23rd of October, or ten days from the sailing of the ship, a case of cholera, ending fatally, occurred in a man twenty-seven years old; a second case followed on the 8th of November, the ship being then in the latitude of Madeira; and now there developed an epidemic which lasted until the ship arrived in the harbour of Rio on the 12th of December, or over a period of nearly six weeks, although there were only sixteen deaths. Among the cabin passengers, officers, and crew, not a single case of sickness occurred.[2]

On the 10th of October, 1871, four days after the last case of cholera

[1] Barrow, in 'Army Med. Reports, 1864,' London, 1866, p. 363.

[2] Kupfer, in 'Viertelj. für gerichtl. Med.,' 1873, Jan., p. 85.

in Stettin, the steamship *Franklin* sailed thence for New York with 486 'tween-deck passengers and 55 cabin passengers and ship's company; at Copenhagen and Christianstad she took on board 129 passengers more, and 12 more of a crew. The cargo consisted of merchandise, and nineteen bales of rags packed in canvas, to which no one had access. The first cases of cholera occurred in children, of whom 9, from one to five years of age, died between the 18th of October (or the tenth day out) and the 28th. On the 28th the first case of sickness and death occurred among the adults, and now the cases came in such rapid succession that by the 6th of November, when the vessel arrived at the quarantine harbour of Halifax, 42 individuals (36 Germans and 6 Danes) had died.[1] On the 6th and 7th of the month, two shore labourers went on board the vessel, the one to take in water and the other to trim coals in the bunkers; they both took cholera at the same time, a few days after, and from the one who had trimmed the coals the disease spread in the first instance to his family, and then in widening circles through the village where he resided (Chezet Cook). "The men Melvin and Lepiere," we read in the account by Lloyd,[2] "employed at the same time in coaling and watering, were exposed for some hours to the poisoned air on board of, or escaping from, a ship in which some 40 deaths had occurred during the previous fortnight, and both took ill the same day, one case proving fatal in twelve hours, and the other communicating the disease to members of his family; all which facts, it must be admitted, clearly prove the propagation of the disease by infection."

It is clear that in these and other epidemics of cholera on board ship,[3] we must admit a continuous series of infections, unless we resort to the incredible assumption of a four or five weeks' incubation-period for the disease, with the object of proving that all those cases of sickness are referable to an infection acquired on shore before sailing,—an assumption which the outbreaks among pilots and shore-labourers at Halifax in 1865 and 1871 render inadmissible.

The epidemics of cholera on board ship are in other respects exactly like epidemics in a house, where we have also a series of cases of infection connected one with another. As in those house-epidemics, the disease on board ship is for the most part associated with some particular deck or

[1] Goeden, in the 'Berl. klin. Wochenschrift.,' 1872, No. 33.

[2] 'Lancet,' 1872, Feb., p. 226 (giving also notes on the events in Halifax itself).

[3] In regard to other similar epidemics of cholera on board ship I refer the reader to the interesting paper of Lawson ('Med. Times and Gaz.,' 1871, Aug., p. 152), which includes, among other cases, a detailed account of the often-quoted epidemic of cholera on board the *Renown*.

section of the vessel, and, further, it has always been those of the passengers most unfavorably situated, or the 'tween-deck passengers, that have suffered most extensively and most severely, if not even exclusively.

§ 121. Diffusion by Human Intercourse. Religious Festivals of the Hindoos.

Even those who deny communicability altogether will hardly question that the *diffusion of cholera*, both within its native habitat and beyond these boundaries, is associated with *human intercourse*;[1] and this conviction cannot be overborne either by the fact that the range of the disease has by no means kept pace always and everywhere with the facilities for intercourse due to improved roads and vehicles, or by the fact that the epidemic does not always follow the great commercial highways, nor even always move forward steadily along the line which it has struck out, but advances by leaps, breaking off suddenly, and as suddenly reappearing at points far distant. It is not easy to decide for each particular case how far this apparently capricious march of the disease is dependent on pure chance, or how far it may be determined by external factors,—by the conditions of atmosphere or of locality. At all events, the introduction of the specific cause, the morbid poison, passes as the first condition for the development of an epidemic of cholera at a particular place, and it is clear that that introduction must in some way or other result from communication. That it is human intercourse which furnishes the media of this communication, is proved on a large scale by the observations on the diffusion of the disease by pilgrimages and military campaigns, both in India and beyond it; while there are other conclusive proofs furnished in innumerable instances from the smaller circles of diffusion.

[1] Balfour, a decided opponent of the doctrine of the contagiousness of cholera, says (in the 'Indian Annals of Med. Sc.,' 1858, Jan., 104) with reference to the epidemic of 1856 in Delhi: "During the epidemic I have seen nothing in any way to lead me to believe that the disease was propagated in this (viz. contagious) manner. . . . It is true that the epidemic travelled to the westward principally by our grand lines of communication."

Earlier observers, such as Coats,[1] Souty,[2] Rogers,[3] and Nash,[4] had already pointed out how important a factor in these visitations were the sacred festivals of the Hindoos, which bring together to the shrines hundreds of thousands of persons from the most remote regions of the country. In 1867 attention was generally attracted to the circumstance, when the disease broke out among the pilgrims collected at Hurdwar and was carried by them for hundreds of miles in all directions. From the report of Cunningham,[5] there is reason to believe that everywhere along the route the first cases of sickness occurred among pilgrims, who, on the appearance of the disease at Hurdwar, had hastened to their homes in wild flight and panic; the disease followed in their train, and spread over fifty-one districts of Hindostan, in which not a single case of cholera had come to observation previously. Further details are given by Beatson[6] for Bengal, and by Monro[7] for the Sirhind district. "The moving mass," says Murray in his general report for the Upper Provinces,[8] "crowded the road in a continuous stream for nearly a week at Meerut, where I remained to watch the course of the disease. This pilgrim stream carried with it cholera, which lined the road with victims, whose funeral pyres studded the surrounding fields, or whose bodies were thrown into the canal or collected by the police and buried. The disease was communicated to the neighbouring towns and villages, and the pilgrims carried it with them to their homes over the whole of Hindostan." The incidents of 1865, when cholera broke out among the Mecca pilgrims, and spread within a few weeks in all directions along the roads taken by them, are still fresh in the recollection of everyone; equally well remembered are the events of the following year, which grew out of the movements of troops in the Austro-German war, and proved so disastrous to Bohemia. "If we weigh the fact," says Kaulich,[9] "that not a single case of cholera occurred in Bohemia before the enemy invaded it, whereas the disease was already prevalent in several Prussian towns and in the Prussian army; if we have regard to the circumstance that cholera appeared in the first instance, and often subsequently, only after the native population had come into contact with Prussian troops suffering from cholera; if we consider, further, that the disease was for the most part carried with great rapidity along the main highways which the invading army followed, that, through a long stretch of

[1] In the 'Report of the Epid. Cholera, &c.,' Bombay, 1819, p. 150.

[2] 'Rapport sur le Cholera observé dans l'Inde en 1829 et 1830, &c.,' Paris, 1832, p. 21.

[3] 'Reports on Asiatic Cholera in the Regiments of the Madras Army from 1828 to 1844,' London, 1848, p. 236.

[4] 'Indian Annals of Med. Sc.,' 1858, Jan., p. 120.

[5] 'Fourth Annual Report of the Sanitary Commissioner, &c.,' Calcutta, 1868.

[6] 'Army Med. Reports for 1866,' London, 1868, p. 335.

[7] Ib., p. 360.

[8] 'Madras Quart. Journ. of Med. Sc.,' 1868, xii, p. 354.

[9] 'Prager Vierteljahrschrift für pract. Heilkd.,' 1875, i, p. 136.

country, the disease did not go beyond the Prussian lines, and that the districts which escaped the invasion were either not attacked by cholera at all, or only long after, and then to a quite insignificant extent; if, finally, we bear in mind that the fortresses shut off from intercourse, as well as the country within range of them, either escaped the disease altogether or were affected only after the blockade was removed, we shall be unable to come to any other conclusion but that cholera was brought into the country with and by the Prussian troops, and was disseminated through it by them. We are also obliged to recognise the fact that the march of cholera followed in general the main arteries of traffic, and that the populous places, where lines of communications met, were the centres from which the disease was scattered abroad." Further, there have been numerous opportunities in India of observing that cholera has progressed by stages corresponding to the marches of troops, of which there is an instance given in the quite recent (1872) account of Lyons from Chittagong.[1]

If the track by which cholera has been imported and transported, has not been discovered in many cases, notwithstanding most careful inquiry, that is explained by the difficulty of watching and controlling the traffic, and especially the traffic in goods, which is unquestionably a more frequent means of conveying the morbid poison than personal intercourse.

Bryden's theory of the "cholera-wave."—A peculiar theory of the *diffusion of cholera by the wind* has lately been developed by Bryden in his doctrine of the "cholera-wave."[2] With reference to cholera in India, Bryden distinguishes, as we have already seen (p. 436), two zones; one, within which the disease is "earthborne," that is, endemic, and the other, in which it is "airborne," that is, epidemic only. The diffusion of cholera throughout its habitat, and beyond it, is, says Bryden, under the influence of the monsoon, and therefore it always follows the direction of that wind; the atmospheric moisture is the proper carrier of the morbid poison, and its geographical distribution extends as far as the moist wind carries it, while dry winds set a limit to the progress of this "cholera-wave." Cholera, says Bryden, is never diffused by human intercourse alone, and the progress of the disease can never be checked by putting a stop to that intercourse. This idea—one may unhesitatingly call it a bold idea—of a "cholera-wave spreading from the Bay of

[1] 'Indian Annals of Med. Sc.,' Jan., p. 391.

[2] 'On the Epidemic Cholera in the Bengal Presidency,' Calcutta, 1869, p. 76.

Bengal to the shores of the Atlantic," has been pretty generally condemned at the hands of Anglo-Indian physicians. Murray[1] declares it to be a "chimera with no more solid foundation than the baseless fabric of a vision;" De Renzy[2] shows it to be absolutely untenable for that part of India for which it was specially designed, viz. for the North-West Provinces; and Macnamara rejects it after critically examining it in detail.[3] Without doubt, says Macnamara, the appearance of cholera in Western Hindostan is connected with the monsoon; "the south-west monsoon would appear to be the indirect cause of the dissemination of cholera over the country, in that it brings with it moisture—a necessary element for the development of the disease—but more especially because it is before this wind that the large fleets of country boats move up the Ganges, conveying men and goods from the home of endemic cholera to be disseminated over the Upper Provinces." But, against the notion that the monsoon, as an air-current, carries with it the germs of cholera from Bengal to the North-West Provinces, there is the fact that the inhabitants of the mountainous parts of Upper Bengal, who feel the influence of that wind, have hitherto escaped the cholera, that the monsoon has never blown the disease from the coast of Burmah or of India to the Andaman Islands, and that in 1817 and 1818, as well as often subsequently, the disease has travelled against the wind. It requires no elaborate proof to show that this monsoon theory, or, in other words, the notion that cholera is spread by the wind, does not at all correspond with the routes taken by the disease beyond India, over Central Asia and Europe, and thence across the ocean to America; nor does it suit the occurrence of the disease at perfectly isolated spots. If, again, the facts do not permit us to ignore that the poison *must* be taken up and suspended in the air so as to enter the human organism with the breath, yet that diffusion of the poison by a current of air (ascending or horizontal) does not hold good at any rate for great distances. This is shown by the narrow limits—often so strikingly

[1] 'Brit. Med. Journ.,' 1874, Jan., p. 73.
[2] 'Report on the Sanit. Administr. of the Punjaub,' Lahore, 1870.
[3] 'History of Asiatic Cholera,' London, 1876, p. 445.

narrow—of cholera foci, outside which there is complete immunity,—by the incidence of the disease upon one town, or one quarter of a town, one street, one house, even one room in a house, without any spreading even to the immediate neighbourhood. This range was estimated by the International Sanitary Conference at Constantinople at a maximum of about 100 metres (110 yards), and by Townsend[1]—much too highly, however—at one English mile.

§ 122. Drinking-Water as a Vehicle of the Poison.

Whether the reception of the poison into the body takes place solely in the way here indicated; whether under other circumstances it may not also reach the organs of digestion with the food; what share, among those media, is taken by the drinking-water in particular; and which way of introduction is the more frequent—these are questions of great practical importance which cannot be decided, in the present state of our knowledge, with the desired amount of certainty. As they lie somewhat outside my theme, I must dismiss them with the single remark that in the controversy which has been carried on about the significance of *drinking water as the vehicle of the cholera poison*, the opposing views have been put forward in a spirit of extreme onesidedness. In particular, the choice that has often been offered between the soil theory or drinking-water theory rests upon an erroneous conception of the question. Without denying the importance of the soil (or its equivalent) for maturing the reproduced morbid poison, one may very well assume that the poison, having acquired potency in the manner indicated, may reach the atmosphere, get deposited on articles of food, or, under certain circumstances, fall into wells and other receptacles of water used for cooking purposes. It might thus reach the organism in various ways. There are many well-known observations which make it probable, at least, that some such connexion subsists between the occurrence of cholera and the use of infected

[1] 'Second Report of the Sanitary Commissioners for the Central Provinces,' Nagpur, 1870, p. 165.

drinking water;[1] but any exact proof that this connexion is a direct one is not forthcoming for the present at any rate, inasmuch as it is always open to us to contend that the drinking water may have been fouled with excrementitious matters, especially the alvine dejecta, that it does not exert a *specific* action, but acts injuriously in a general way through the putrid matter that it contains, and is calculated merely to heighten the predisposition of the individual for the specific attack of sickness. The inquiries that may be directed in the future to this highly-important question should be made to follow a somewhat more rational plan than hitherto. They should not be content with determining the amount of decomposition-products of organic matter in water, or with detecting lower organisms in the suspected sample; but they should first of all ascertain what effect this water exerted upon the population in question before the disease broke out and after it had ceased. With the chemical and physical aids at our disposal we have not yet succeeded in making out a *specific* (*choleraic*) *infection of water;* and if a suspected water be proved by chemistry or by the microscope to be impure, that does not of itself justify us in concluding certainly as to infection, any more than the negative discovery precludes from so doing. The specifically injurious action of a sample of drinking water can only be inferred if exact proofs are forthcoming that cholera sickness has been caused by partaking of it.

The epidemiological and pathological relations of cholera to miliary fever have been discussed in treating of the latter disease (§ 33).

[1] See especially the following works: Snow, 'On the Mode of Communication of Cholera,' London, 1855 (German, by Quedlinburg, 1856); Farr, in the 'Report on the Cholera Epidemic of 1866 in England,' London, 1868, pp. 87 ff; Ballot, in the 'Nederl. Tijdschrift voor Genesk.,' 1868, pt. ii, p. 173, and in the 'Med. Times and Gaz.,' 1869, May, p. 459, and June, p. 426; 'Rapport van de Commissie tot Onderzoek van Drinkwater in Verband met de Verspreiding van Cholera,' s'Gravenhage, 1868-1869; Förster, 'Die Verbreitung der Cholera durch die Brunnen,' Breslau, 1873, and in the 'Zeitschrift für Epidemiologie,' 1874, i, p. 81.

CHAPTER X.

PLAGUE.

(Bubo-Plague.)

§ 123. Ancient, Mediæval, and Modern Epidemics: the Black Death.

The history of the plague may be followed into remote antiquity, and, with a certain measure of certainty, even as far as the end of the third or the beginning of the second century of the pre-Christian era. In the 44th book of the 'Collectanea' of Oribasius,[1] discovered by Cardinal Mai, there is a note, taken from the writings of Rufus, to the effect that the contemporaries of Dionysios[2] make mention of a certain disease as "pestilentes bubones maxime letales et acuti, qui maxime circa Libyam et Ægyptum et Syriam observantur." Rufus then gives a description of this disease according to observations made of it in Libya by two physicians, Dioscorides and Poseidonios, who lived in Alexandria about the Christian era; and the description leaves no doubt as to what it was.[3] There are no other certain

[1] First published in 'Classic. auct. e. Vaticanis cod.,' t. iv, *c.* 7; afterwards commented on by Osann ('De loco Rufi Ephesii med. apud Oribasium servato, sive de peste libyca disp.,' Giessen, 1833), and by Bussemaker ('Diss. exhibens librum xliv collect. med. Oribasii,' Groning, 1835).

[2] This Dionysios, with the surname ὁ κόρτος (native of Cyrta), or ὁ κυρτὸς (the hunchback), is mentioned by Hermippus, in the 'Lives of Eminent Men.' Hermippus lived about the 125th Olympiad, *i.e.* 280—277 B.C., and Dionysios must have been either contemporary with him or before him (compare Daremberg in Prus, 'Rapport sur la peste, etc.,' Paris, 1846, 239).

[3] The passage, as translated by Bussemaker (p. 33), runs: "Dioscorides autem et Posidonius plurima de hac re enarrant in libro de peste, quæ eorum ætate in Libya adfuit; illi autem accedere dixerunt febrem acutam, dolorem, perturbationem totius corporis et delirium et bubonum apparitionem magnorum et durorum, qui in suppurationem non transiebant, non solum in solitis locis verum et in poplitibus et cubitis; quamvis illic omnino tales inflammationes non solent

references to the plague in the writings of earlier or contemporary physicians, or in those of the later period of antiquity,[1] or in the historical writings of the chroniclers of that period.[2] It is not until the sixth century that we meet with authentic descriptions of the bubo-plague, the first epidemiological records of it occurring in the accounts, given by numerous writers,[3] of the severe pestilence which spread over the whole Roman Empire of the East and West, and even far beyond the limits of the empire, in the time of Justinian. According to report, the pestilence started in 542 A.D. from lower Egypt (particularly from Pelusium), and, taking the direction, on the one hand, of the North Coast of Africa, and, on the other hand, of Palestine and Syria, it came to Europe at a time of profound disturbance and confusion in political and social affairs, and overran it in the course of the following year "to the ends of the habitable world." This pandemic is estimated by contemporary writers to have lasted fifty to sixty years, during which the disease broke out anew at many places, and

observari; fortasse autem buboniformis morbus Hippocratis constitutionem dictam indicat; aderit autem nonnunquam et in genitalibus talis bubo, uti et ulcus pestilens et febris quam pestilentem dicunt; plerumque epidemica talia sunt, ita ut communia sint omnibus ætatibus et constitutionibus in nonnullis anni temporibus præcipue occurrentia."

[1] Hippocrates several times ('Aphor.,' iv, § 55, ed. Littré, iv, 521, and 'Epid.,' lib. iii, § 55, e. c. v, 108), mentions the occurrence of buboes in severe febrile diseases (*ὁι ἐπὶ βουβῶσι πυρετοὶ πάντες κακοὶ, πλὴν τῶν ἐφημέρων*). It is doubtful, as Rufus has pointed out (*vide supra*), whether the buboes of the plague are actually meant here; Littré also, in his interesting exposition ('Oeuvr. d' Hippocrate,' ii, p. 584), expresses himself with great caution on the point. These cursory references of Hippocrates are repeated in Aretæus ('De causis acut. morb.,' lib. ii, cap. 3, ed. Kühn, p. 38), and in Galen ('De different. febr.,' lib. i, cap. 7, ed. Kühn, vii, 296, 'De method. med. ad Glauconem,' lib. ii, cap. 2, e. c. xi, 6, in 'Hippocr. epid. lib. ii Commentat.' iii, § 10, e. c. xvii, A. 410, and 'Comment. in Hippocr. Aphor.,' iv, § 55, e. c. xvii, B. 733).

[2] It is very questionable whether the pestilence of the 3rd century described by Cyprian and others, was plague; it is clear that they are treating of a mixture of various kinds of disease, as in the case of the epidemic at Athens during the Peloponesian war in the description of Thucydides. It is possible that plague may have been one of them, but it is not proved.

[3] Procopius, 'De bello Pers.,' ii, c. 22; Evagrius, 'Hist. Eccles.,' iv, c. 29; Agathias, 'Hist.,' v, c. 9; Gregory of Tours, 'Hist. Franc.,' iv, c. 5, 31, vi, c. 14, 33, ix, c. 22, x, c. 1, 25; Warnefrid, 'De gest. Longobard,' ii, c. 4; Sigbert, 'Chron. in Rer. Germ. gest.,' i, 523.

wrought the most frightful devastation wherever it appeared. In the words of Warnefrid, it depopulated towns, turned the country into a desert, and made the habitations of men to become the haunts of wild beasts. It cannot be denied that this pandemic, like former ones, included several diseases, notably smallpox, which were prevalent at the same time; but the bubo-plague takes at any rate a foremost place among them. Even if there could be any doubt, after the terms "pestis inguinaria" or "glandularia," used by the chroniclers to designate it, the descriptions given by Procopius, Evagrius, and Agathias will enable us to decipher the characters of the plague without difficulty, although they are certainly obscured in various ways by the admixture of other diseases.

It is impossible, as we have seen, to decide whether this outbreak of plague in the second half of the sixth century was the first general diffusion of the disease on European soil, or whether it had been epidemic there before, and if so, to what extent. What is certain is that this outbreak gave it a firm hold in Europe, and that it kept its dominion there for more than a thousand years.

The history of pestilence in the middle ages is full of notices by the chroniclers of severe "pests;" and the conjecture that plague had figured prominently among these pestilences is justified by a reference to the history of the disease in the sixteenth and seventeeth centuries, and is made all the more probable by the fact that many of these outbreaks are expressly designated by the authorities with such names as "clades inguinaria," or "glandularia," or "pestis bubonica." But our knowlege of the facts relating to the bubo-plague in those times goes no farther than that; the chroniclers mostly confine themselves to giving the date when the pestilence occurred, and the number of the victims, which they often unquestionably exaggerated. Still less do we learn anything from the medical writers of the middle ages, who thought they had done their duty when they furnished subtle speculations as to the influence of the stars or unusual appearances in the heavens, on the origin of the sickness. Nowhere in these writings, apart from the records that belong properly to epidemiology, is

there any detailed account of the disease; and the little that there is about it goes to prove that they held fast to the old notion of "pestis" or λοιμός, thereby confusing various severe kinds of epidemic disease, and that they did not get to understand the specific nature of the plague. There is one only of the epidemics of plague in the Middle Ages that has arrested the attention of the chroniclers, poets, and physicians of those days; and that interest was awakened by the enormous diffusion that it reached over the whole of the then known world, by its victims reckoned in millions, and by the shock to the framework of society which it brought with it and left behind it. This disastrous pestilence, known everywhere under the name of the *Black Death*, as one of the great events in the world's history, has fixed the attention of writers in a high degree, and has been thought worthy to be painted in minutest details and in the most vivid colours.[1]

I shall reserve for a subsequent part of this chapter, the more particular discussion of the character of this epidemic of plague, distinguished as it was by a prominent affection of the lungs. For the present I shall give merely the historical facts as to the outbreak and progress of the epidemic, with a glance at the elucidation which the history has gained at the hands of Hecker,[2] and more recently of Haeser.[3]

The starting point of the pestilence is placed by contemporary writers with one accord in Eastern Asia,[4] whence it spread in a westerly

[1] The most important references to this pestilence by physicians occur in Guido de Cauliaco, 'Chirurgia Tract.,' ii, cap. 5 (Lugd., 1572, 113); Chalin de Vinario, 'De peste libri iii' (Lugd., 1552), and Dionysius Colle in Joh. Colle, 'Medicina pract.' (Pisauri, 1617, 570, reprinted in Haeser, 'Hist. pathol. Untersuchungen,' ii, 525). Among the accounts in chronicles, special attention is due to the descriptions of the jurist Gabr. de Mussis (in 'Haeser's, Archiv.' 1842, ii, 26), and, among poetical sketches, to the artistically perfect account of the events in Boccaccio's 'Decamerone,' Giorn. i, Introd.

[2] 'Der schwarze Tod, &c.,' Berlin, 1832. (Somewhat amplified from later sources, in my collected edition of Hecker's 'Schriften über die Volkskrankheiten des Mittelalters,' Berlin, 1865, 19.)

[3] In the 3rd ed. of his 'Lehrbuch der Geschichte der Medicin und epidemischen Krankheiten,' Jena, 1875-82, Bd. iii, 97.

[4] Most writers place the original habitat of the black death in the mysterious country of Cathay (to which the name of Chitai, still in use in Russia, corresponds) or China. Fracastori, to whose opinion I shall return in the course of the inquiry, locates it in the region of the Ganges.

direction, most probably by various routes, over the countries of Asia Minor to the North Coast of Africa and to Europe. There is no information as to the year in which the epidemic began, or as to the speed with which it travelled, up to the time of showing itself on the frontier between Asia and Europe. According to De Mussis, who was an eye-witness of the Crimean outbreak, the pestilence was prevalent in 1346 in Sarmatia (Tanais) and Caffa (now Feodosia) in the Crimea, and it appears to have reached Arabia and Egypt about the same time. In the beginning of 1347, it was imported into Constantinople, and in the autumn of that year it came from the Crimea by shipping to several ports on the Adriatic and Mediterranean coasts of Italy, as well as to the South of France (Marseilles). From the coast it spread in 1348 into the interior of those countries, reaching Spain at the same time, and the Netherlands, England, and the Scandinavian kingdoms a little later. The year after, it was in Switzerland, Germany, Poland, and Russia; so that, by the end of that year (1349) or the beginning of the next, the whole continent of Europe and most of the islands[1] adjoining it, had been overrun by the plague. There is little known of a trustworthy kind as to the area of diffusion of this epidemic of plague in extra-European countries; according to the current saying, no part of the then known world escaped it. The loss of life by the disease is certainly unexampled in the history of pestilence. Although most statements of the number of victims are unreliable, yet there is information as to the mortality given for a few small centres, which may be trusted, and may serve as an approximate measure of the mortality in general. Hecker puts the whole number of those who perished in the epidemic in Europe at the figure—not too high—of twenty-five millions, or about one-fourth of the then population of our division of the globe.

It is difficult for us to decide how long the plague had continued in the characteristic form of the Black Death; its extinction in some places followed after a duration of a few months, in other places not until a whole year or even several years; but we can form no safe opinion on the nature of the "pests" that sprang up anew shortly after, inasmuch as nearly all that we know of them comes to us from the records of chroniclers. This much, however, is certain, that among these pestilences, the bubo-plague again took a leading place,[2] and that even in later epidemics, the pheno-

[1] From Norway it was imported into the Faröe Islands, the Orkneys, and the Shetlands. Iceland, according to Schleisner (l. c., 56), remained exempt from the Black Death; but there is no doubt that Greenland was visited by it, and some historians are inclined to date the decline of culture in that country from this pestilence (see Ilmoni, 'Nordens sjukdoms historia,' i, 132).

[2] In the years 1360-61, 1363, 1371-72, 1379-83, and 1399-1400. On the

mena distinctive of the Black Death occurred with so great frequency, that the chroniclers could not help being reminded of that disease.

The history of pestilence in the fifteenth century is hardly less rich in notices of "epidemics of plague" than that of the fourteenth; and although that designation still included other severe forms of spreading sickness, yet a large proportion of the epidemics were undoubtedly the bubo-plague.[1] An important subject of inquiry and discussion among the physicians who had happened to make closer acquaintance with the nosological character of the disease since the outbreak of the Black Death, was its origin and mode of spreading, with special reference to contagiousness and to the use of embargos and quarantines which had come into vogue since the Black Death. Besides these questions, they made the type and course of the plague a subject of close observation; and therewith they took an important step towards a more accurate knowledge of the various diseases of peoples and towards a resolution of the great generic term "pest" into its several elements. One of the first fruits of this improved diagnosis shows itself about the end of the fifteenth century, in the recognition, for the first time by the professional world, of exanthematic typhus, which had certainly played a prominent part among the "pestilences of antiquity and the Middle Ages."

Throughout the sixteenth century, the plague was a permanent form of disease on the continent of Europe, and that century furnishes us with a considerable number of good epidemiological records of it.[2] It seems as if scarcely a year

epidemics of plague in those years and in the century following, see Corradi, 'Annali delle epidemie occorse in Italia,' i, 218 ff; Villalba, 'Epidemiologia española,' i, 87 ff; Ilmoni, l. c. i, 185 ff; Richter, 'Geschichte der Medicin in Russland,' i, 227 ff.

[1] It was especially widespread in Europe in 1400-1402 (in the latter year for the first time in Iceland), and later in 1437-40, 1448-50, 1463, 1482-83, and 1493-94. Between these dates come numerous epidemics limited to particular regions, or to one or more countries.

[2] The most numerous and best epidemiological records of the plague in the sixteenth century come to us, as we might have expected, from Italy, which was then the centre of learning in general. Among these writings, so far as I know them, the most noteworthy appear to me to be Massa's 'Raggionamento sopra le infirmità . . . del presente anno 1555,' Venet., 1556, the 'De peste . . . libri iv,'

passed without the plague raising its head and spreading, sometimes as an epidemic over smaller areas, at other times in a continuous progress from country to country with the character of a pandemic. During the first two-thirds of the seventeenth century, we still meet with it over an equally wide area and equally often. But in the last thirty years of that century, the plague was observed to be retreating gradually from the soil of Europe, and at the beginning of the eighteenth century it became still more certain that a turning-point had been reached in the fatality from plague which had hitherto hung so heavily over the populations of Europe. Only twice afterwards did the pestilence become at all widely diffused in the western and central regions of the Continent. From the middle of the eighteenth century, only the south-eastern parts were a permanent seat of the disease; from these it frequently made incursions northwards, but it hardly ever got beyond the Balkan peninsula and the countries immediately adjoining. Since the beginning of the present century, it is only in the region last mentioned that plague has been epidemic from time to time, on the last occasion in 1841; and, if we except the slight epidemic of the winter of 1878-79 in the Government of Astrakhan, it then vanished completely from the soil of Europe.

Among the countries of Europe to be exempted earliest from the visitations of plague, were the *Scandinavian kingdoms*. There was an

of De Oddis (Venet., 1570), the 'Decem problemata de peste' of De Bonagentibus (Venet., 1556), and the 'De origine et causa pestis Patavinæ anni 1555,' of Landus (Venet., 1555)—all relating to the epidemics in Venice and Padua in 1555; the account of the epidemics of 1575 and 1577 in those cities by Mercurialis ('De peste in universum, præsertim vero de Veneta et Patavina prælectiones,' Basil, 1577); Massaria's 'De peste libri ii,' relating to Vicenza (Venet., 1597, and in his 'Opp.,' Lugd., 1669, 485 ff); and the account by Ingrassia for Palermo, 'Informatione del pestifero e contagioso morbo . . . nel anno 1575-1576,' Palermo, 1576. Among the German writings on the plague there are three that deserve special mention: the small tract of Vochs, 'De pestilentia anni præsentis, &c.,' 1537, on the epidemic in Germany during the first ten years of the century; Böckel's 'De peste quæ Hamburgum civitatem a. 1565 adflixit,' Henricopoli, 1577; and Rod. de Castro's 'Tract. de natura et causa pestis, &c.' (Hamb., 1596), on the Hamburg epidemics of 1565 and 1596. Of French accounts I know only the 'De peste liber' of Joubert (Lugd., 1567), a compilation of no great importance relating to the plague at Lyons in 1564; and of Spanish, the most excellent work of Boccangelino, 'De febribus morbisque malignis et pestilentia . . . liber' (Madrid, 1604), on the plague in Spain in 1599.

importation of it into Denmark and Sweden in the beginning of the eighteenth century; but, apart from that, the last epidemic in the one country was in 1654,[1] and in the other in 1657.[2] The last considerable epidemics in *Italy* were in 1656 and 1657;[3] it reappeared once towards the end of the same century, in 1691 at Conversano and Monopoli (Province of Bari), having been imported from Cattaro, but it did not spread beyond those places.[4] *England* was visited by the plague for the last time in 1665;[5] in *Ireland*, which had never suffered very much from it, the last epidemic was in 1650. After the severe pestilence of 1664-66 in the *Netherlands* and *Belgium*,[6] the disease has reappeared only once, so far as I know, at Bruges in 1669, and then only to a limited extent.[7] In Switzerland the last epidemic of plague was in 1667-68 at Basel[8] and in a few communes of the Canton Zürich.[9] The same years saw also the last outbreak in *France*,[10] (not counting the appearance of the plague in Provence in 1720) as well as throughout the greater part of Western *Germany*; whereas in the eastern and south-eastern regions of that country, it occurred subsequently in 1679-81, and for the last time in the first years of the following century (*vide infra*). Finally, in *Spain*, the last records of plague are for the years 1677-81, in which the disease spread over a considerable part of the southern and south-western littoral.[11] *Turkey*,[12] as we have seen, and the countries immediately adjoining it, were almost the only seat of plague in Europe during the eighteenth century. Twice only did the pestilence extend its epidemic ravages beyond that narrow area. The first occasion was from 1707 to 1714, when it issued on the one hand from Russia to overrun Silesia, Prussia, and Pomerania, spreading thence to Holstein and Brunswick and severely attacking several places in Denmark

[1] See Bartholinus, 'Cista med.' Havn., 175.

[2] Account in the 'Vecko-Skrift for Läkare,' viii, 72.

[3] See Corradi, 'Annali delle epid. occorse in Italia,' iii, 185.

[4] Ib., 273.

[5] Hodges, 'Λοιμολογία, or an Historical Account of the Plague in London, &c.,' Lond., 1721.

[6] Piens, 'Tract. de febribus, &c.,' Colon. Allobr., 1689, 6, 416, 480; see also Israels, 'Nederl. Tijdschr. voor Geneesk.,' 1873, ii, 14.

[7] 'Chronyke van Vlaenderen,' iii, 733.

[8] Verzascha, 'Centur. I observ. medicarum,' Basil, 1677, obs. 53-64, p. 112, *seq.*

[9] See Meyer-Ahrens in Hufeland's 'Journ. der Heilkd.,' 1839, lxxxix, Heft 3, 60.

[10] Lepecq de la Cloture, 'Med. topogr. d. Normandie,' from the French, Stendal, 1794, 307.

[11] Villalba, 'Epidemiol. española,' i, 123, *seq.*

[12] According to information which is tolerably reliable, the plague was epidemic in Turkey, and specially in Constantinople, at least eighteen times during the eighteenth century (1702-3, 1716, 1730, 1738 to 1739, 1741, 1747, 1749-50, 1753-54, 1755-57, 1762-64, 1768, 1770-71, 1773, 1777, 1783, 1790-91, 1795-96, 1797). For the period from 1800 to 1830, there are references to epidemics in 1800-1802, 1812-14, 1819, 1826-27, and 1829.

(Elsinore and Copenhagen) and in Sweden (Karlskrona, Upsala, and Stockholm), and, on the other hand, from Prague in 1713, visiting Austria, Styria, and the south-east of Bavaria.[1] The second of the two epidemics affected the Mediterranean seaboard of France. The disease was introduced into Marseilles in 1720 by a vessel from Syria; it spread over a great part of Provence, and did not die out until 1722.[2] For the remaining seventy years of the century, we find reference to only one outbreak of plague in Western Europe, namely, at Messina in 1743; the last attack there had been in 1624, and this time it was imported by a vessel from the Levant.[3] In the nineteenth century, there have been no considerable epidemics of plague in Europe, excepting in the Balkan peninsula and the countries adjoining it. Isolated cases of it occurred in Malta in 1813,[4] in the small Italian seaport of Noja in 1815, and in the Balearic Islands, especially Majorca, in 1820,[5] having been probably imported from Barbary.

As the area of the plague in Europe has become narrower, and the channels of its diffusion have come out clearer, the

[1] The following writings deserve to be specially mentioned among the extremely abundant literature relating to this epidemic: Kanold, 'Einiger Medicorum Sendschreiben von der a. 1708 in Preussen grassirten Pestilenz, &c.,' Brsl., 1711; Gottwald, 'Memoriale loimicum, &c.,' Dantisci, 1710; Stoeckel, 'Anmerk. bei der Pest, 1709, in Danzig,' Danz., 1710; Gohl, 'Histor. pestis, &c.,' Bresl., 1709; Diderich, 'Untersuchung der Seuche, welche in Hamburg a. 1708 eingerissen,' Augsb., 1714; Mahr, in the 'Arch. für Gesch. der med.,' ii, 261, and 'Die Pest in Glückstadt im Jahre 1712,' Kiel, 1879; Böttticher, 'Morbor. malignor. imprimis pestis explicatis, &c.,' Hamburg, 1713; Mansa, 'Pesten i Helsingör og Kjöbenhavn, 1710 og 1711,' Kjöbenh., 1842; v. Rosenstein, 'Tal om pesten,' Stockholm, 1712; Werloschnig et Loigk, 'Historia pestis, &c.,' Styr. 1715 (very complete account of the pestilence in Austria and at Regensburg); Weitenweber, 'Mittheil. über die Pest in Prag 1713 und 1714,' Prag, 1852. Concerning the plague in Silesia, see also Lorinser, 'Die Pest des Orients, &c.,' Berlin, 1837, 437 ff.

[2] There is a very copious literature also in connexion with this epidemic, the most noteworthy memoirs being Chicoyneau's 'Traité des causes, des accidens et de la cure de la peste, &c.,' Paris, 1744, and d'Antrecheau's 'Relation de la peste . . . de Toulon, &c.,' Paris, 1756.

[3] Turriano, 'Memoria istor. del contagio della città di Messina, &c.,' Napoli, 1745; Testa, 'Relazione istor. della peste. . . . a Messina, &c.,' Palermo, 1745. See Corradi, 'Annali,' iv, 122.

[4] Faulkner, 'Edin. Med. and Surg. Journ.,' 1814, April, p. 137, and 'Treatise on the Plague, &c.,' Lond., 1820; Tully, 'History of the Plague, &c.' (Lond., 1821), Agra, 1837; Calvert, 'Med.-Chir. Trans.,' 1815, vi, 1. A succession of cases occurred again in Malta in 1815, but they did not amount to an epidemic (Hennen, 'Sketches of the Medical Topogr. of the Mediterranean,' Lond., 1830).

[5] Schönberg, 'Ueber die Pest zu Noja, &c.,' Nürnb., 1818; D' Onofrio, 'Detaglio istorico della peste di Noja, &c.,' Napoli, 1817; Morea, 'Storia della peste di Noja,' Napoli, 1817. Cf. Corradi, 'Annali,' iv, 646.

more decidedly does *Turkey* stand revealed as almost the sole point of departure in Europe for every inroad of the pestilence. Even in some of the great epidemics of plague in the seventeenth century, it was possible to follow the track of the disease from the east, towards the northern, central, and western parts of the Continent. That route was still more decided in two severe epidemics at the beginning of the eighteenth century, of which we have spoken; and it was very obvious in subsequent times down to the extinction of the plague in Europe about the year 1840, that is to say, within the period when the disease existed nowhere out of Turkey, except in the countries of the Lower Danube and in Southern Russia.

Thus in 1738-39, owing to the state of war in Wallachia, the plague reached the Ukraine,[1] and then Hungary and Transylvania.[2] In 1756 it spread from Turkey to Bucharest, Podolia, and Cronstadt. In 1770 there was again a conflict between the Russian and Turkish armies in Wallachia, and a severe epidemic of plague broke out among the troops, which overran part of Southern Russia, penetrated even to Moscow,[3] and paid a visit to Transylvania.[4] There was an importation from Turkey to Kherson and to Dalmatia in 1783, to Transylvania[5] in 1786, to Syrmia[6] in 1795, and to Volhynia[7] in 1798. The outbreak of 1811 in Turkey gave rise to a severe epidemic which spread, during that year and the next, by way of Odessa[8] to Podolia and the Crimea, and to Wallachia[9] and Transylvania[10]. During the years following, there were several outbreaks in the Austrian Military Frontier,[11] with which the above-mentioned epidemics at Noja and in Malta were connected. The latest considerable outbreak in the East of Europe, was also a consequence of war; it was in 1828-29, when the Russian and Turkish forces

[1] Schreiber, 'Observ. et cogitata de pestilentia, &c.,' Berol., 1744.

[2] Hammer, 'Geschichte der Pest . . 1738-40 im Temeswarer Banate, &c.,' Temeswar, 1839.

[3] Orræus, 'Descriptio pestis, &c.,' Petrop., 1784; Samoilowitz, 'Mém. sur la peste, &c.,' Par., 1783; Mertens, 'Traité de la peste,' Vienne, 1784.

[4] Chenot, 'Historia pestis Transsylvanicæ, &c.,' Bud., 1799.

[5] Lange, 'Rudimenta doctrinæ de peste, &c.,' Offenb., 1791; Neustädter, 'Die Pest im Burzenlande, &c.,' Hermannstadt, 1793.

[6] V. Schraud, 'Geschichte der Pest in Syrmien,' 2 Theile, Pesth, 1801.

[7] Minderer, in 'Hufeland's Journ. der Heilkd.,' 1806, xxiv, St. 2, 1.

[8] Account in the 'Salzb. med.-chirurg. Ztg.,' 1814, ii, Nos. 29, 30.

[9] Grohmann, 'Beobachtungen über die Pest zu Bukarest,' Wien, 1816.

[10] Plecker, 'Oest. med. Jahrb.,' 1834, Nst. F., vi, 211.

[11] Pfisterer, 'Beobacht. und Abhandl. oester. Aerzte,' 1821, ii, 213; ib., 269; Roch, ib., 1819, i, 1.

came into collision in Wallachia,[1] the pestilence getting imported into Odessa[2] and Cronstadt[3]. In the very same years, there was an epidemic in Greece, which had been free from plague for more than a century; it was introduced by Egyptian troops who had been sent to replace others[4]. Since 1830, the plague has been epidemic only once in European countries outside Turkey, namely, in 1837, at two points, the Greek island of Poros[5] and Odessa,[6] but only to a slight extent in either.

In Turkey itself, we meet with the disease in 1834, 1836, 1837, and 1839; it appears to have been in Constantinople once more in 1841, but with the extinction of that outbreak, there has been no more plague even in Turkey,[7] down to the present day.

Whereas in Europe, the whole mainland, together with the insular territories adjoining, has been repeatedly since the sixth to the middle of the nineteenth century, the scene of epidemics of plague of various extent, on *African* soil the area of the disease never extended beyond the northern coast belt; and it was *Egypt*, as in Europe it was Turkey, that here formed the starting point of the numerous epidemics which overran the Berber States during those centuries.

An enumeration of the epidemics in Egypt has no particular interest, and there is the less occasion to undertake it, as the information about them in past centuries is extremely meagre and not always trust-

[1] Czetyrkin, 'Die Pest in der russischen Armee,' Berl., 1837; Seidlitz, Petersenn and Rinck, in the 'Abhandl. pract. Aerzte zu St. Petersburg,' 5 Samml., 1835, 44, 135, 169, 203; Witt, 'Ueber die Eigenthümlichkeit des Klimas der Wallachei und Moldau,' Dorpat, 1844.

[2] Wagner, in 'Hufeland's Journ.,' 1830, lxx, St. 2, 109.

[3] Plecker, l. c.

[4] Millingen, 'Lond. Med. Gaz.,' 1831, May, viii, 298; Gosse, 'Relation de la peste en Grèce, &c.', Par., 1838; Bobillier, 'Journ. gén. de méd.,' 1829, tom. cvi, 401.

[5] Wibmer, in 'Rohatzsch's Zeitschr. f. Chir.,' 1842, Nr. 35—38; Link, in 'Hufeland's Journ.,' 1839, lxxxviii, St. 4, p. 5.

[6] Andrejewsky, 'Die Pest in Odessa,' from the Russian, Berl., 1839.

[7] For the history of the plague in Turkey during the present century see: Valli, 'Sulla peste di Constantinopoli del 1803,' Mantova, 1805; Legrand, 'Nouv. Journ. de méd.,' 1818, ii, 288; Schuller, in 'Hecker's Annalen der Heilkd.,' 1831, xix, 164; Moreau, 'Transact. méd.,' 1830, i, 418; Röser, 'Württemb. med. Correspdbl.,' 1835, iv, 51; Cholet, 'Mém. sur la peste,' Paris, 1836 (see also Prus, 'Rapp. sur la peste,' 624); Brayer, 'Neuf années à Constantinople,' Paris, 1836; Müller, 'Oester. med. Jahrb.,' 1841, ii, 32, 191; Pezzoni, 'Lettre au sujet des accidents de peste, &c.,' Constantinople, 1841; Thirk, in 'Oest. med. Wochenschr.,' 1846, 820, 849, and in 'Oest. med. Jahrb.,' 1847, ii, 148.

worthy. It must suffice to mention here that there had been twenty-one epidemics in the period from 1783 to 1844, viz. in 1783, 85, 88, 90, 94, 96-97, 99, 1800-1803, 1810-12, 1815, 18, 24, 25, 28, 31, 33-34, 35, 36-37, 38, 40-41, and 1843-44. Several of these embraced a period of two years or more; between the several epidemics, the free intervals were short (two to five years); the last epidemic of plague in Egypt was in 1843 and 1844, and, since then, not a single case has been observed.[1]

The disease had frequently spread, as we have remarked, from Lower Egypt westwards, through the Barbary States of *Tripoli*, *Tunis*, and *Algiers*, and sometimes even to Morocco. The records are too scanty to let us judge with any certainty of the frequency of the disease in these countries.

According to a calculation of Berbrugger,[2] the plague had been epidemic in Algiers about thirty times from 1552 to 1784, and several of these epidemics lasted more than a year. Since 1784, five epidemics have been observed in Algiers, in 1786-87, 1793-97, 1799, 1816-21, and 1836-37; but the last was confined to the Date-country, into which it had been imported from Tripoli.[3] Tunis and Tripoli must always have had a part in these Algerian epidemics, inasmuch as the natives of Algiers were agreed in saying that the pestilence had always come to them from the eastward. On the other hand, it does not seem to have penetrated often to *Morocco*;[4] only six epidemics are mentioned in the period from 1678 to 1817, viz. those of 1678, 1741, 1750, 1787, 1799, and 1818-19. Since the extinction of the 1837 epidemic in Tunis and Algiers, and that of 1818-19 in Morocco, there has been absolutely no

[1] On the plague in Egypt, see the following: Prosper Alpinus, 'Medicina Ægyptiorum,' Lugd. Bat., 1719, 59; Frank, 'De peste, dysenteria, et ophthalmia ægypt,' Vienna, 1820; Wolmar, 'Abhandl. über die Pest.,' Berl., 1827; Pugnet, 'Mem. sur les fièvres de mauvais caractère du Levant,' Lyon, 1804; Desgenettes, 'Hist. méd. de l'armée d'Orient.,' Paris, 1802; Lagasquie, 'Revue méd.,' 1829, Nov. 207, 1834, i, 39, 171, 338; Pariset, 'Mém. sur les causes de la peste,' Paris, 1837; Assalini, 'Reflessioni sopra la peste d'Egitto,' Torino, An. ix; Pruner, 'Die Krankheiten des Orients,' Erlang., 1847, p. 387, and 'Ist denn die Pest wirklich ein ansteckendes Uebel?' Münch., 1839; Clot-Bey, 'De la peste observée en Egypte,' Par., 1840; Aubert, 'De la peste ou typhus d'Orient,' Par., 1840; Bulard, 'Ueber die orientalische Pest,' from the French, Leipz., 1840; Lefèvre, 'Essai critique sur la peste,' Stuttg., 1840; Fischer, in 'Jahrb. d. ärztl. Vereins zu München,' ii, 90; Iken in 'Casper's Wochenschr. für die Heilkd.,' 1837, No. 47, 745; Rossi, 'Relazione della pestilenza . . . in Damiata,' Livorno, 1841.

[2] In Prus, 'Rapport sur la peste et les quarantaines,' Paris, 1846, 259.

[3] Guyon, 'Gaz. méd. de Paris,' 1838, No. 49, 1839, No. 32.

[4] Nion in Prus, 'Rapport,' 620. On the epidemic of 1818-19, see Gråberg di Hemsö, 'Lettera sulla peste di Tangeri,' Genove, 1820, and the same in 'Annal. univ. di med.,' 1820, xiv, 304, and in 'Svensk. Läk. Sällsk. Handl.,' 1819, vi, 187.

plague in those countries. It is only in Tripoli that it has reappeared of recent years, on two occasions, of which more in the sequel.

Almost all the authorities agree, concerning the diffusion of the plague in Egypt, that Lower Egypt has always been the headquarters of the disease, that it has not unfrequently extended to Upper Egypt, but has always reached its limit in Assuan, never going higher than the First Cataract, and never entering Nubia.[1]

Abyssinia also has remained untroubled by the plague, and the same is true of the whole *East* and *West Coasts of Africa*, including *Senegambia* and the *Cape Colony*; so that, as we have said, the area of the disease on the African continent has been strictly limited to the territories of the Northern Coast.

The history of bubo-plague on *Asiatic* soil has a particular and direct interest at the present time. So far as the scanty and not always reliable information about the behaviour of the disease in past centuries enables us to form an opinion, *Syria* takes a foremost place as regards the frequency of epidemics;[2] the localities that have suffered most have been a few places on the coast, such as Tripolis, Jaffa, and Beyrout, and the inland district of Aleppo. From 1773 to 1843, it appears to have occurred thirteen times, the last outbreaks being in 1832-39[3] and 1841,[4] and it has not shown itself there in subsequent years.

The pestilence has been observed almost as often on the

[1] Aubert, l. c. 101. Wolmar (l. c. 11), it is true, gives an account of an epidemic of plague which raged in 1696 in Nubia and "the Kingdom of Dongola," but he has neglected to show that the pestilence was really plague. Equally undeserving of trust are the statements of Petherik ('Egypt, the Soudan, and Central Africa,' London, 1861, 332), who alleges that the plague was once prevalent in Khartoum; of Tutschek ('Oestr. med. Wochenschr.,' 1846, 1207), who relates, on an obviously unreliable authority, that there had been a destructive "pest" in Kordofan seventeen years before his arrival, or in 1828, which had swept away whole tribes around Tumale; and of Baker ('The Albert Nyanza, &c.,' Lond., 1866, ii, 300, 333, 340), who met with a severe pestilence in Khartoum on his return from Lake Nyanza, in the end of 1864; he designates it "plague or malignant typhus," but, in his description of the appearances of the disease, there is not a single symptom characteristic of plague.

[2] See Russell (Patrick), 'Treatise of the Plague,' 4to Lond., 1791; Lasperanza, in Prus, 'Rapport,' 487.

[3] Grassi, in Prus, 'Rapport,' 403; Granet, ib., 503.

[4] Robertson, 'Edin. Med. and Surg. Journ.,' 1844, Oct., 330, 1845, Oct., 345.

coast of *Asia Minor*. From 1771 to 1837, there are reports of twelve epidemics, the last of which were in 1833,[1] and 1837-39.[2] The history of plague in *Caucasia* is wanting in reliable information for previous centuries; but, if we may judge from what we know of recent times, the disease must have been very widely epidemic. The first trustworthy notice[3] is for the year 1798, in which there was a severe epidemic in Georgia; it broke out afresh in the autumn of 1802 at Tiflis, and continued to crop up at one part of the country or another for sixteen years, or until 1818. Whether there may not have been outbreaks of plague there subsequently, I shall not undertake to decide. For *Armenia* also, the history of plague cannot be followed with certainty beyond the beginning of the present century.[4] Since that date, it has occurred in 1805, 1807-8, 1811, 1824-25, 1827-28, 1839, and last in 1841. In contrast to these regions of Asia Minor hitherto mentioned, which had been permanent seats of the plague down to about 1850, *Mesopotamia* and *Persia* had seen very few epidemics of it up to the latter date.[5] For Mesopotamia, we have only a single reference in epidemiology to plague in previous centuries, the outbreak at Bagdad in 1596; and it has occurred only thrice in the period from 1750 to 1842, in 1773, 1800-1801, and 1830-34. From the very thorough inquiries of Tholozan for Persia, we have information of epidemics in 1571-75, 1596, 1617, 1636 (limited to one place), 1685-86, 1725, 1757, 1760-61, 1773, 1797 (also limited), and 1829-35; from the last date until the outbreak in Kurdistan in 1863, nothing had been heard of the plague in Persia. It is worthy of note, that while, in all the countries of Asia Minor already mentioned, the pestilence extended more or less over the whole country, on Persian soil it was practically confined, in all these twelve epidemics, to the north-west of the country, particularly to

[1] Marpurgo, ib., 608.

[2] Floquin, in 'Memor. della medicina contempor.,' 1838, Nov., 55; Tholozan, 'Comp. rend. de l'Acad.,' 1875, lxxxi, 123, and 'Gaz. méd. de Paris,' 1875, 393, 419.

[3] Tholozan, l. c.

[4] Lachèze in Prus, 'Rapport,' 293.

[5] See particularly Tholozan, 'Histoire de la peste bubonique en Perse et en Mésopotamie,' II. Mémoires, Paris, 1874.

the province of Azerbijan (including Kurdistan), while the northern provinces of Gilan, Mazanderan and Khorassan have been visited only once, and the whole interior of the country, as well as the south and east, not at all. *Arabia* is another of the countries where the plague has been frequent, as Pruner tells us, in recent times; but down to the close of the period of which we are now speaking (the year 1842), only two epidemics (in 1815 and 1832) are recorded. On both occasions, the disease was very destructive along the coast region, in Yembo and Jeddah, and inland as far as Mecca. There have been epidemics there subsequent to 1850, of which more in the sequel.

Until quite recently, everyone considered it settled that Persia was the eastern limit of the area of plague on Asiatic soil, beyond which, during the last five centuries at least, it had never penetrated. But the latest observations in India and China have completely upset that opinion. They have furnished us with extremely interesting facts as to great centres of plague in the interior regions of Eastern Asia; and this addition to our knowledge is all the more calculated to arrest the attention, for the reason that these centres belong not only to the past, but remain unimpaired down to the present day. Taken along with the latest outbreaks of plague in various parts of Nearer Asia, this discovery of plague-centres in India and China is likely to place the history of plague in a somewhat different light from that of our earlier and more superficial views.

The first trustworthy information on the occurrence of *plague in India* dates from the year 1815.[1]

In May of that year, the disease broke out in the Island of Cutch, in a district where there had been a famine shortly before; next year it

[1] See the accounts by Gilder, 'Transact. of the Bombay Med. Soc.,' 1838, i, 190; McAdam, ib., 183; Whyte, ib., 155; Glen, 'Quart. Journ. of the Calcutta Med. Soc.,' 1837, i, 433. Macpherson ('Annals of Cholera,' Lond., 1872, 112) quotes from an Indian chronicle the following notice of a malignant disease that had been prevalent towards the end of the seventeenth century: "A fever had prevailed for some years both in the Deccan and in Guzerat. It consisted of a slight swelling under the ears, or in the armpit or groin, attended with inflamed eyes and severe fever. It generally proved fatal in a few hours." This is suggestive of plague; and it is further noteworthy that the disease occurred in that very region which formed the seat of the first epidemic of plague in India of which we have any certain knowledge.

was in the Gujerat States, and from them it spread in a westerly direction to Sind, the capital of which suffered a severe visitation in November, 1816. It was in 1817 that the disease reached the nearest British territory at Dollera, whence it continued its progress until the end of the year over the districts of Dunduka and Limri, and next year as far as the shores of the Rann of Cutch. In 1819, Buriad was attacked, and Dollera for the second time in 1820, the disease penetrating at the same time to the district of Ahmedabad. It was not until 1821, and after a duration of six years, that the epidemic died out completely. A second outbreak took place in 1836, and this time the Rajpootana States were the seat of it. It broke out first in July, 1836, at Pali,[1] the entrepot of the trade between the North-West Provinces and the coast, and quickly spread over an area of thirty miles radius from the town. In October it reached Jhodpore, the capital of the State of Marwar, and it continued its progress in an easterly direction over the range of hills into the State of Mewar, in which it attacked thirty-two places in the course of the following year, the British cantonments at Nassirabad escaping through a rigorous quarantine. Towards the end of the year, it broke out anew at Pali, and did not die out until the spring of 1838.

These facts, showing the prevalence of plague from 1815 to 1838 in the low country of Hindostan, are borne out by two notices which have just come to my knowledge, and have been hitherto quite overlooked. The one is that of Col. Skinner,[2] who states that in 1828-29 a disease absolutely like Pali plague ("the same in all its features, even glandular swellings"), and with the same malignancy, had been prevalent in Hansi, in the Province of Delhi. The other is that of Guthrie,[3] who remarks that, while the plague was at Pali, there was a pestilence observed over the whole country round Bareilly (Rohilkund), which had precisely the same characters.

Along with these isolated outbreaks of plague in Hindostan, and perhaps connected with them, there has been an endemic centre of plague *on the southern slopes of the Himalaya* and in

[1] See Irvine, 'Quart. Journ. of the Calcutta Med. Soc.,' 1837, i, 241; notices in the same, 245—55; Sheriff, ib., 429; Russell, ib., 445; Maclean, ib., 17, and 'Ind. Journ. of Med. Sc.,' 1836, n. s., i, 617, 1837; n. s., ii, 380; Forbes, 'Transact. of the Bombay Med. Soc.,' 1839, ii, 1 (also his thesis, 'On the Nature and History of Plague as observed in the N.-W. Provinces of India,' Edin., 1840); Ranken, 'Report on the Malignant Fever called the Pali Plague,' Calcutta, 1838.

[2] 'India Journ. of Med. Sc.,' n. s., ii, 389.

[3] Ib.

the provinces of Kumaon and Gharwal, the existence of which can be traced back with certainty to the year 1823.[1] It is said to have shown itself first in 1823 in several villages situated above the snow-line in the districts of Nagpore and Budaon,[2] but in 1834-37 it became more widely diffused, following the course of the Pindar. In 1846-47 several villages were attacked, situated at the sources of the Ramgunga at a height of more than 10,000 feet, as well as certain districts in the province of Kumaon. From 1847 to 1853 the disease does not seem to have quite left the wide territory which it had taken possession of; and we should perhaps not greatly err, in connecting with this general diffusion of the plague, the epidemic of 1835, previously mentioned, in the province of Rohilkund to the south of Kumaon, as well as the outbreak of a pest-like sickness, a "typhous fever, and, in addition, buboes in the groins, axillæ, and neck," which we learn from a recent paper of Murray's[3] to have been prevalent in 1832 in the mountainous district inhabited by the tribe of the Jussafzai, forty miles to the north of Peshawur. There can be no doubt that in all those epidemics it is well-marked plague that we have to deal with. The same is true, also, of a pestilential centre in China, of an extent at present undetermined, the existence of which was brought to notice first in some recent publications[4], although it is unquestionably of older date. The seat of the pestilence is the mountain valleys of Yun-nan; when and how it came there cannot be learned from the state-

[1] The fact that the natives of the affected districts were already provided with a special name for the disease—"mahamurree," afterwards "bola"=boil—at the time when this centre of plague first became known, seems to prove that its origin was of much older date there. Planck is also of that opinion. Compare the notice, quoted above from Macpherson, of a pestilence in Gujerat about the end of the seventeenth century, characterised by glandular swellings.

[2] Compare Webb, 'Pathologia Indica,' Lond., 1848, 212; Renny, 'Med. Report on the Mahamurree in Gurhwal in 1849-50,' Agra, 1851; Pearson and Francis, 'Indian Annals of Med. Sc.,' 1854, April, 627; Bird, 'Med. Times and Gaz.,' 1854, May, 498; Planck, in 'Papers relating to the Modern History of Plague' (Parliamentary Papers), Lond., 1879, p. 48.

[3] 'Med. Times and Gaz.,' 1878, June, 597.

[4] Baber, in 'Parliamentary Papers, China,' No. 3 (1878), pp. 22, 23, reprinted in 'Papers relating to Modern History of Plague,' p. 39; and Manson, in 'China Imperial Maritime Customs Medical Reports,' 1878, quoted in 'Med. Times and Gaz.,' 1878, Nov., 576.

ments of the natives. Several high-placed mandarins declare that it was imported from Burmah, and that there was no sign of it in the central and eastern parts of the province previous to the outbreak of the revolution; others say that it had been observed still earlier in the extreme west of the province, in the neighbourhood of Tali-foo the capital, that it had spread eastwards, doubtless owing to the military operations, and had ultimately overrun the whole province. We have accurate data about the prevalence of the disease, and the frightful ravages that it committed, for the years 1871-73; nothing certain is known of it in subsequent years, but we may infer from the reports that it was at least not extinguished as late as 1879.

§ 124. Recent Outbreaks in Asia Minor, &c.

While in Hindostan the plague has continued from its first appearance (or its first becoming known) in 1815 through all the subsequent years, as a more or less widely-diffused pestilence, it has entirely died out, since the end of the epidemics of 1841-44, in all those regions of Asia Minor, Africa, and Europe which had been up to that time its permanent habitat; that is to say, Syria, Asia Minor, Egypt, and Turkey in Europe. On the other hand, since 1850-70 new centres of plague have sprung up in other parts of Nearer Asia and of Africa, which had previously been little affected—in Arabia, Mesopotamia, Persia, and the coast of Tripoli, and this development has made, to some extent, a new era in the history of the plague.

The first in the order of time of these latest outbreaks of plague, is the outbreak in 1853 in *Arabia*, where it had last occurred in 1832. Here it is clearly the mountainous district of Assir, belonging to North Yemen, that forms the seat of the disease. Just as it spread thence in 1853, apparently over the greater part of Arabia, so on its reappearance there in 1874, it penetrated to within four days' march of Mecca;[1] a third time it broke out at the same place in 1879,[2]

[1] There is an account of both epidemics by Dickson, in 'Papers relating to the History of the Plague,' p. 16, and one by Buez, in 'Gaz. hebdom. de méd.,' 1875, 52, according to the information of Medical Inspector Dr. Pasqua.

[2] 'Lancet,' 1880, January, p. 67.

but there is nothing certain known hitherto of the extent of that epidemic.

Next in order of time comes the outbreak of 1858 on the coast of Tripoli, where there had been no plague since 1843.

The pestilence arose in April among a tribe of Arabs living in the vicinity of the port of Benghazi[1]; it spread gradually over most of the province, and died out in June, 1859. There was a second outbreak at the same place in April, 1874, but it was limited to a few tribes of Arabs inhabiting the Cyrenaic plateau, and it ended in July; among a population of 734 souls, there were 533 cases of sickness and 208 deaths.[2]

The third in the recent series is the outbreak in Persia, where the disease had not been seen since 1835.

It showed itself first in the autumn of 1863 in several villages in the vicinity of Maku (Persian Kurdistan), which town it afterwards attacked, dying out in the beginning of the following year.[3] A second outbreak in the same locality followed in December, 1870, on the table-land lying to the south-east of Lake Urumiah; the winter brought it within narrow compass, but in spring and summer it spread to fourteen (or seventeen) villages situated at a height of 6000 to 7000 feet, and close to the Turkish frontier; it ceased in September, 1871,[4] after causing 891 deaths among a population of about 7000. A third epidemic occurred in Persia in 1876, in Schooster, a large town of Khuzistan, and a number of villages near it; it was introduced by a caravan which had arrived from the infected districts of Mesopotamia, and it carried off some 2500 victims (of which 1800 were in the town of Schooster), coming to an end in July.[5] Towards the end of the same year (1876) it broke out in two villages of the province of Khorassan, situated at a height of 1000 metres (3300 feet), and distant about 100 miles from the south-eastern corner of the Caspian. Connected with that outbreak there is the epidemic in March, 1877, at Resht, the capital of the province of Gilan, situated at the south-western corner of the Caspian, about five miles from the coast; it spread thence over many villages in the neighbourhood, and died out in the beginning of next year (1878). At the same time the plague is said to have shown itself again in one of the districts of Persian Kurdistan, where it had been in 1871.[6]

[1] Bartoletti and Fauvel, in 'Rec. des travaux du Comité consultatif d'hyg. publ. de France,' iv, 151.

[2] Arnaud, 'Essai sur la peste de Benghasi en 1874.' Constantin., 1875.

[3] According to the reports of the English medical officers in 'Papers, &c.,' p. 7. The Persian authorities appear to have received no notice of this epidemic.

[4] Tholozan, 'Gaz. méd. de Paris,' 1871, 588, and in his 'Histoire de la peste bubonique en Perse,' Paris, 1874, 31; Castaldi, 'La peste dans le Kurdistan Persan,' Constantinople, 1872.

[5] Millingen, in 'Papers,' p. 27.

[6] Tholozan, 'Compt. rend.,' 1877, lxxxv, No. 8, 432; 'Papers relating to the History of Plague,' 37-38, 44-47.

The fourth and most considerable of the recent foci of plague is in Mesopotamia, in the plain of the Tigris and Euphrates, where the disease had been epidemic last in 1834.

Plague broke out first in the winter of 1866-67, among the Arab tribes inhabiting the marshy level of Hindieh on the west of the Euphrates; but both there and at other points in Mesopotamia it is said that there had been, during several years previously, many isolated cases suspected to be plague.[1] The epidemic was confined to five villages, in which 300 persons died, and it came to an end in June.[2] Six years after (1873) it reappeared in the same locality, and this time grew to an epidemic which lasted five years, and overran the greater part of the country in wider and wider diffusion.[3] The beginning of this epidemic falls in December, 1873, when the disease showed itself in Dagarra, a district that suffered more than any other except Divanieh; a certain number of deaths from plague occurred also in Hillah, Nedjef, Kerbela, and other places. In June it appeared to have died out, after carrying off 4000 victims among a population of 80,000 in the localities affected; but in winter (1874-75) it broke out afresh, spreading this time southwards through Divanieh; it again remitted during the summer and autumn (1875), but once more revived in autumn and invaded the plain of the Tigris, although it had been for those three years practically confined to the Euphrates valley. In March, 1876, it appeared in Bagdad. Nothing certain is known of the range of the pestilence in that year; the number of its victims is estimated at 20,000, but that number is probably much too small. There was again an intermission during the summer and autumn, but in November (1876) it developed into a new epidemic. In June, 1877, the plague in Mesopotamia appears to have become extinct altogether; there has been at least, no news of plague from these parts since that date, although that does not preclude its continued existence.

Plainly connected with these recent epidemics in Mesopotamia and Persia, although the connexion cannot be traced in detail, is the outbreak of plague in the south-east of

[1] This fact, given by Naranzi, is not quite free from doubt, but it is deserving of every consideration in a historical respect, as we shall see afterwards when we come to sporadic instances of plague.

[2] Dickson, 'Transact. of the Epidemiol. Soc.,' iii, 143; Colvill, 'Lancet,' 1867, July, and 'Transact. of the Bombay Med. Soc.,' 1871, 49; Tholozan, 'Une épidémie de peste en Mésopotamie en 1867,' Par., 1867; Naranzi, 'Gaz. méd. de l'Orient,' 1868, Juill., 57, Août, 72; Barozzi, ib., 1869, Mars.

[3] Castaldi, 'La peste dans l'Irak-Arabi en 1873-74.' Constantinople, 1875; Tholozan, 'Compt. rend.,' 1877, vol. 85, 432; Lavitzianos, 'Bull. de l'Acad. de méd. de Paris,' 1878, No. 45; Colvill, 'Lancet,' 1876, May, 681; Dickson, 'Brit. Med. Journ.,' 1878, March, 339; Adler, 'Allg. Wien. med. Ztg.,' 1879, Nos. 4—11; Wortabet, 'Edin. Med. Journ.,' 1879, Sept., p. 222, Oct., p. 297.

Russia, at a few places near the Volga in the Government of Astrakhan. The preceding occurrence of the plague there, probably after importation from the Caucasus, had been in 1808; that epidemic had travelled up the Volga as far as the Government of Saratov, but to so limited an extent that the total of deaths due to it hardly exceeded one hundred.[1] In this latest outbreak, which took place in September or October, 1878, the epidemic was confined to the village of Vetlianka on the right bank of the Volga, while in five other villages cases of sickness and death occurred more sporadically. It was extinguished in January, 1879, having caused a mortality of about 600.[2]

§ 125. Relation to Climate.

Until not very long ago it passed as an uncontested doctrine that the territory of plague found its natural limit at the *tropic* zone; and that opinion was based principally on the fact that the disease, notwithstanding its many hundred years' existence in Lower and Middle Egypt, had never penetrated into the tropical regions of that country (or to Nubia),[3] and never into the adjacent country of Abyssinia. The recent experiences in Arabia and India—in the latter the incidents occurred from 1815 to 1821—have overthrown that doctrine, or have taught us, at least, that it cannot be maintained in its integrity. It cannot be doubted that *climatic influences*, and above all the lowness of the *thermometer*, are full of significance for the breaking out of the disease and its growth to an epidemic; but the purport of all experience hitherto does not justify us in placing the cause of tropical Africa's exemption from plague solely in the temperature peculiar to it, or in putting that etiological factor at the head of the list with as much exclusiveness as formerly. The following data as to the relation of epidemics of plague to seasons and

[1] Milhausen, 'Abhandl. der Gesellsch. pract. Aerzte zu St. Petersburg,' vii, 39.

[2] Hirsch and Sommerbrodt, 'Mittheilungen über die Pest-epidemie im Winter, 1878 bis 1879,' &c., Berlin, 1880; Zuber, 'La peste du gouvernement d'Astrakhan,' &c., Paris, 1880.

[3] See the note on p. 506.

weather will serve to keep these proportionate assignments within their due bounds.

It is in *Egypt* that the dependence of epidemic plague upon season and weather comes out most definitely. The law laid down by Prosper Alpinus,[1] that the disease appears in Egypt in autumn and dies out in June, has been accepted by all subsequent observers,[2] although with some limitations; and the statistics of mortality for the last years of the plague in Alexandria afford a complete proof of it.

Table of Deaths from Plague in Alexandria, 1834-43, in the several months.

	1834.	1835.	1836.	1837.	1838.	1839.	1840.	1841.	1842.	1843.	Total.
January .	—	242	20	17	—	—	13	32	5	—	329
February .	—	951	35	3	—	—	27	66	19	1	1112
March . .	—	4459	20	20	—	—	179	246	26	2	4952
April . .	—	2016	8	31	36	—	400	407	46	2	2936
May . .	—	592	49	34	71	27	396	515	82	33	1799
June . .	—	48	19	10	74	20	180	212	62	20	547
July . .	1	—	15	6	39	1	71	67	10	6	216
August. .	48	1	17	3	4	—	6	17	3	1	100
September	—	3	4	3	2	—	1	2	—	—	15
October .	—	3	11	—	—	—	—	4	—	—	18
November .	38	9	12	—	2	—	—	1	1	—	63
December .	150	19	14	—	1	—	10	1	—	—	195

In *Syria* the extinction of the epidemic in the plains takes place usually in June or July, at Aleppo in July or August, while in the mountainous regions it has continued not unfrequently all through the summer and autumn and even into the winter.[3] On the coast of *Asia Minor*

[1] L. c. 70: "Pestis Cayri atque in omnibus locis Ægypti invadere eos populos solet ineunte Septembri mense usque ad Junium . . . . Junio vero mense, qualiscumque et quantacumque sit ibi pestilentia, solo primam Cancri partem ingredienti, omnino tollitur, quod multis plane divinum esse non immerito videtur. Sed quod etiam valde mirabile creditur, omnia supellectilia pestifero contagio infecta tunc nullum contagii effectum in eam gentem edunt, ita ut tunc ea urbs in tutissimo atque tranquillissimo statu reducatur, ex summe morbeso."

[2] Wolmar 12, Pruner 419, Aubert Roche, and others. The beginning of the epidemic varies between September and January, sometimes even later. On the other hand its extinction falls with great constancy in the end of June or beginning of July; Frank gives it, for Alexandria, exactly on St. John's Day (24th June), and for Cairo at the summer solstice (21st June); but as Russell remarks (i, 320), these dates are not to be taken altogether literally.

[3] Russell, Robertson.

(Smyrna, Trebizond) the plague season has usually been from February to August,[1] and in *Turkey in Europe* from June to October. In *Mesopotamia* the epidemics from 1867 to 1877 nearly all reached their height between March and May. In *Persia* the epidemic of 1870-71 (in Kurdistan) came to a head in winter and spring, that of 1876 (in Schooster) in May and June, and that of 1877 (in Resht) during the summer. In *Yun-nan* it broke out in May, was prevalent to a considerable extent during the summer (rainy season), and reached its height as the rains ceased and the dry season set in. In *India* no particular association with season could be traced in the epidemics of 1815-21 and 1836-38, nor during the period when the disease was prevalent in Kumaon.[2] In *Arabia* the epidemic of 1832 (on the west coast) was in May, but that of 1874 (on the table-land of Assir) was from April to September, the greatest intensity falling in July and August. In *Algiers* the plague season has mostly been spring and autumn. But on European soil outside Turkey, it has been summer and autumn; of 88 epidemics at various places during this and the previous century, of which we have accurate accounts, 17 began in winter, 22 in spring, 26 in summer, and 23 in autumn, while the culmination was 8 times in winter, 13 times in spring, 48 times in summer, and 26 times in autumn.

We may draw from this the general conclusion that a moderately high temperature favours the epidemic prevalence of plague, but that extremes of heat and cold mostly cause the extinction of the epidemic, or are little favorable to its breaking out. There are, however, many exceptions to this rule; and the statement of Pruner, that the disease cannot exist as an epidemic except between the degrees of 17° and 22° R. (Robertson gives 12·5° to 21·5° or 67° to 87° Fahr.) may be set aside as unfounded.

Those who would explain the exemption from plague, which Upper Egypt has always enjoyed, by its weather, and particularly by its high temperature, are naturally brought to a pause when they reflect that the disease has been prevalent in subtropical and tropical parts of Asia and Africa under a temperature which has not only equalled but exceeded that of the spring in Upper Egypt; and if plague may exist as an epidemic in Middle Egypt under a temperature of from 25° to 30° R. (from autumn to spring), it is not inconceivable that it might thrive in the Nubian winter and spring with a temperature of 18° to 20°. Examples of the outbreak or persistence of plague under extreme heat are furnished: by the epidemic of 1735 in Smyrna (where the heat was so excessive at the time of the plague that persons who left the

[1] Aubert-Roche, Thirk, Lachèze.

[2] In Gharwal it broke out in 1849 towards the end of the rainy season, and lasted till December; while in 1850 it appeared in March and had become general as early as May.

town for the neighbouring villages died of sunstroke on the way),[1] by that of Malta in 1812, of Algiers in 1813,[2] of Greece[3] in 1828, of Roumelia[4] in 1837, of Resht in 1877, and of Kumaon in 1850.[5] But plague has been epidemic in extreme cold still more often than in extreme heat; as, for example, at Rome in 1656-57,[6] at Marienburg[7] and at Stockholm[8] in 1710-11, in Transylvania in 1708-9,[9] in Provence[10] in 1720-21, in the Ukraine[11] in 1738, in Greece[12] in 1727-28, in Roumelia[13] in 1737-38, in Kurdistan[14] and in Kumaon in 1849.[15]

[1] Mackenzie, 'Philos. Transact.,' 1753, vol. 47, 384.

[2] Berbrugger, in Prus, 'Rapport,' l. c.

[3] Gosse, 61: "The heat in July was excessive, with the warm winds from the south. In the vicinity of Argos two persons died from the effects of the great heat."

[4] Müller, 38: "The moist warmth of spring gave place to the protracted heat and drought of summer. . . . The Egyptian tradition that the dew which falls at the end of June will destroy the matter of the plague, was not borne out by the events in Roumelia in 1837 . . . . The sultry heat of August gave the plague a new lease of life."

[5] "Mahamurree," says Rennie, "has prevailed in temperatures beyond which it is known that the plague is destroyed or suspended in Europe and Africa. . . . At Bhungdar on the 17th May, the thermometer in the shade stood at 83° F. maximum in the day; at Mason . . . . it may be affirmed that the heat was much greater even a month earlier. . . . At Deghat . . . the maximum thermometer on the 19th of May was 95° at three p.m."

[6] A Castro, p. 11: "Romæ etiam inter rigores hiemales . . . . non desinit glisscere."

[7] Erdl, 'Ephemer. med.-phys. Acad. Leopold. Cent.,' v and vi, obs. 130, p. 247.

[8] Rosenstein, 'Tal om pesten,' Stockh., 1772-4.

[9] Chenot, 'Abhandl. von der Pest.,' Dresden, 1776, p. 54. "The plague that invaded Transylvania in 1708 lasted through the severest cold of the following winter."

[10] The severe winter (snow and ice) imposed no limits on the epidemic; it was raging with extraordinary malignancy during that season at Arles, Toulon, and various other places.

[11] Schreiber, p. 5: "Mense Julio usque in hiemem intensissimam, immo alicubi locorum et per eam integram saeviit morbus popularis."

[12] Gosse, p. 71: "During the cōld and moist winter, when the men were under canvas or in the bivouacs, the plague spread throughout the cantonments of Ibrahim's army."

[13] Muller, l. c.: "During the winter, when the temperature fell at times to − 13° R., the pestilence became extinct at some places, but at others it continued the whole winter through."

[14] 'Papers,' p. 12: "Whilst the population [at elevations of 5000 to 6000 feet] were thus snowed up, built in by the snow in the midst of indescribable filth, plague became active among the infected communities and almost extinguished several."

[15] Rennie says: "One spot [among the villages attacked] has been named, as high as 10,000 feet above the sea, which elevation must give a constant temperature low enough to check the plague, whereas the report is that Mahamurree has been as virulent in such a climate as elsewhere."

Several of those who have observed the plague in Egypt (Alpinus, Wolmar, Larrey,[1] Pruner, Penay,[2] and others) had laid particular weight on the injurious influence of the *kamsin* upon the continuance and severity of the epidemic, and they have trusted especially to the observed fact that an extinction of the epidemic is associated with the cessation of that desert wind, and with the setting in of the so-called "etesian" winds from the north and north-east. It is highly improbable that the *kamsin* possesses "specific" properties which might cause an increase in the extent and severity of the epidemic; the influence of this wind would be manifested most decidedly in the elevated temperature, and we should thus account for the contradiction which these observers find themselves in, with their views about high temperature being a restraining influence on the pestilence. But this factor in the production of plague should not be rated too highly, inasmuch as the pestilence not unfrequently appears when the desert wind is failing, or after it has given place to northerly winds. Clot-Bey[3] is of this opinion, and he adds that he had never himself observed an increase of the epidemic while the *kamsin* was blowing, but rather a diminution of it; and Mackenzie remarks that he had not made out any favorable effect of the "etesian" (north-east) winds in Constantinople on the course of the epidemic.

We should find it equally difficult to accept the opinion, advocated especially by Pugnet,[4] that *a high degree of atmospheric moisture*, taken by itself or in conjunction with high temperature, affords a material condition for the development of plague to an epidemic, and that the overflow of the Nile, which has been so often adduced as a factor in the etiology, becomes a cause of the disease, not through the saturation of the ground, but through the increased moisture rising into the atmosphere from the ground. Against this view we have the fact that many epidemics of plague in Europe during former centuries, and in Asia in recent times, have

[1] 'Med.-chir. Denkwürdigkeiten, &c.' From the French. Leipzig, 1813, 111.

[2] In Prus, 'Rapport,' 539.

[3] Op. cit., 219.

[4] Pugnet says (202): "The plague in Egypt is always an affair of a humid atmosphere."

broken out and run their course under circumstances of place and time which have altogether excluded the influence of a high degree of atmospheric moisture.

§ 126. Relation to Soil and Altitude.

Many of the earlier inquiries into the genesis of plague are subject to the general defect that their authors had attended principally or almost exclusively to the circumstances under which the disease arose in Egypt, Turkey, and other parts of the Levant. Hence the doctrine, which has been in full currency until recently, that the plague, in becoming epidemic, is associated essentially with low, alluvial, and copiously watered levels. Many facts from the history of pestilence in past times tell against that notion; and the observations that have been made since the fresh outbreak of the disease, in so far as we may conclude from them hitherto, place in a very questionable light the doctrine that *particular states of the soil* have some significance for the pathogenesis.

As regards the *immunity of elevated regions*, the idea is supported by a few facts;[1] but against it there is, on the one hand, a series of earlier experiences of the disease in Syria and in the Atlas in Algiers at elevations of 1000 metres (3200 feet), and in Armenia (Erzeroum) at 2000 metres, and, on the other hand, and even more decidedly, the recent discovery of the disease in the mountain districts of Persia (Kurdistan), Arabia (Assir), China, and India, where the pestilence extends its sway to heights of 3000 metres and upwards. So that, at the present day, there is some reason for regarding an elevated locality as more predisposed to plague than a low plain.[2]

[1] A well-known instance is the constant immunity, in spite of repeated importations, of the Abul-Daghun mountain, situate five leagues from Constantinople, and 1800 feet high. The same is told of a hill in Malta, which has, for that reason, acquired the name of "Safi," or "the healthy;" but Hennen has observed that when the plague broke out in Malta in 1812, the locality of Zebug, which is just as high and as airy, had a severe visitation.

[2] Compare Tholozan, "Du développement de la peste dans les pays montagneux et sur les hauts plateaux de l'Europe, de l'Afrique et de l'Asie," in the 'Compt. rend.,' 1873, vol. 77, p. 107.

That the *geological character of the soil* is without any influence on epidemic occurrence of the plague is shown by a glance at the area of the disease, which includes equally the chalk soil of Aleppo, the trachyte of the Armenian Highlands, the limestone of the Ionian Islands, and the alluvial and diluvial coasts of the three continents of the Old World. But there has been particular stress laid on the *physical characters of the soil*, on its permeability and its hygroscopic properties, the Nile Valley being considered to be in these respects a classical example of a plague-soil. Almost all the observers of the plague in Egypt agree in saying that certain processes in the soil, when it has first been saturated by the overflow of the Nile and then left to dry by the waters running off, are directly concerned in the development of the disease; and this conviction has been strengthened by the fact, which has been ascertained on many occasions, that the plague has extended just as far as the inundation has reached, and that there has been immunity beyond those limits. This opinion, which was maintained by Prosper Alpinus and by many physicians after him, and has been widely accepted, has found additional support in the recent outbreaks in Irak-Arabi along the swampy banks of the Euphrates and Tigris. There have certainly been some Egyptian authorities who have raised objections to this theory, which we cannot dismiss peremptorily. Thus Bulard[1] points out that there is an overflow of the Nile every year, and yet the land has been free from plague for years together; that the many villages which lie like islands in the midst of the flooded plain, are by no means the earliest or the most inveterate seats of the disease; that the fellahs, although they work in this flooded ground covered with Nile mud as with a crust, and are exposed therefore to its direct effects, do not suffer from plague either sooner or more severely than the urban population, and that sometimes, indeed, few or no cases of sickness occur among them while the pestilence is raging in the adjacent towns. Against the theory, further, there are many facts from the history of plague outside Egypt. It has often been epidemic in various parts of Europe, especially in England, the south-west of Germany, and in Scandinavian

[1] Op. cit., 35.

countries, where the state of the soil in no wise corresponded to those conditions of the plague-soil as defined. It has often broken out in years which have been remarkable for the want of rain, when there could be no thought of a saturation and subsequent drying of the ground; and, again, it has been prevalent at a time when the ground was frozen and covered with snow, when the changes in its deeper strata, accordingly, could not exert any influence upon the exterior. But the experience of the most recent outbreaks supplies us with arguments of scarcely less weight against the assumption that the development of plague to an epidemic is absolutely dependent on that state of the ground of which we have spoken. The plague in Kurdistan, prevalent throughout the severest part of the winter, and at an elevation of 2000 metres (6500 feet), had its seat on a dry, stony, and barren soil of limestone. On the plateau of Assir, it spread over a fruitful region quite free from any large water-courses or marshes, while the swampy plain forming the declivity was exempt. In India it has been prevalent equally in the damp valleys and jungle-covered slopes of the Himalaya, and on the rocky and dry soil of Kattiwar or the sandy and barren plain of Ahmedabad and Mewar. Tholozan[1] has, therefore, reason for saying: "The points at which the bubonic fever may originate are found equally in mountainous districts at a great height and on a dry and non-alluvial soil, and in the low and damp plains of Mesopotamia." But there are still many observations which tell in favour of a dependence of epidemic plague on certain local circumstances. It is very often confined within a narrow range which it does not exceed, notwithstanding the free and often busy communication with the neighbourhood. Not unfrequently there are a few points terribly smitten in the midst of a wide expanse of country, while at many other localities the cases may be only sporadic for a great length of time. Those places, again, which escape in one epidemic, may become the scene of severe outbreaks on other occasions. The only possible explanation of this remarkable feature in the mode of diffusion of the disease, must be to assume certain local peculiarities, either permanent or occasional, which favour the

[1] 'Peste en Perse,' 42.

genesis of the disease or make it possible, and whose absence precludes its development to an epidemic. A clear insight into the nature of these local peculiarities is not attainable in the present state of our knowledge. But we have at least reason to place them rather in hygienic deficiencies than in any characteristic qualities of the soil; although even that is a point of view which is far from presenting a complete solution of the problem.

§ 127. Intimately Connected with Want and Filth.

There is no point in the etiology of plague about which observers at all times and in every place have been so entirely in agreement, as that the origin and diffusion of the disease are closely connected with the injurious influences of a *defective hygiene*, and particularly with domestic misery. Almost all the authorities on epidemics of plague in Europe during past centuries point to the accumulation of filth in the houses and in the streets, to defective disposal of fæcal matters and other animal excreta, to overcrowding and insufficient ventilation of dwellings, and the like, as a real means of fostering the pestilence; they all urge the removal of these noxious influences as the most important principle of prophylaxis, and they all remark that the reason why the plague has mostly, and sometimes exclusively, attacked the poorer part of the population, is that among them the defects of social well-being are most felt.

"The first outbreak of plague," says Orraeus[1] of the epidemic of 1770 in Jassy and Moscow, "was everywhere in squalid tenements and dwellings, where the air was moist, warm, and impure; when it showed itself in houses of a better class it was extinguished without much trouble. . . . Even when it was at its height, if perchance it found its way into houses of the larger and better sort, it was never known to attain great virulence, and it was quickly suppressed by due precaution . . . The plague spent its fury upon the people, and comparatively few people in a better station of life were carried off by it. This was the uniform experience in the epidemic of which I write, as it had been in others." The same opinion is expressed by Diemerbroeck[2] for the plague of 1635 at Nymwegen, by those who saw the epidemic of 1680 at

[1] 'Descriptio pestis,' p. 51.

[2] Op. cit., lib. ii, cap. 3.

Leipzig,[1] by Böttticher[2] for that of 1711 at Copenhagen, by Stöckel[3] for the Danzig epidemic of 1709, by Lange[4] for the plague in Transylvania in 1786, by Minderer[5] for that of 1798 in Volhynia, and by Gosse[6] for the epidemic of 1828-29 in Greece. Muratori,[7] in summing up the experiences of the Italian epidemics, says: "Perciò nelle contrade più stratte, e ricolme di poveri abitanti, entrato che vi sia il male, si vede in poco tempo una spaventosa desolazione; e le città più popolate restano a proporzion più afflitte, che l'altre men popolate."

But the same conclusions are come to by all who have observed the disease[8] in Egypt, Syria, Asia Minor, and Turkey in Europe. In like manner all the information that has reached us regarding the latest outbreaks in India, Mesopotamia, and Persia is agreed in saying that nothing has fostered so much—and in India still fosters—the epidemic (and endemic) prevalence of plague as the frightful misery in all that relates to dwellings, clothing, and the like, and the total absence of all consideration for rational hygiene. The opinion is also unanimous, that the classes most exposed to such injurious influences have suffered from plague most severely.

Rennie, in his sketch of the social condition of the villages in Gharwal that were attacked by the pestilence first, and with greatest severity, says: "The filth is everywhere—in their villages, their houses, and their persons. . . Their dwellings are generally low, and ill ventilated except through their bad construction; and the advantage to the natives in other parts of India of living in the open air is lost to the villagers of Gharwal, from the necessity of their crowding together for mutual warmth and shelter against the inclemency of the weather." A similar picture of the unhygienic conditions of that region is drawn by Francis and by Pearson; and Murray, in a recent paper on plague in India, says: "All the medical officers concur in the dissemination being essentially promoted by crowding, bad ventilation, and defective sanitary

[1] 'Leipziger Pestschade, &c.,' Altenb., 1681, cap. 9.

[2] Op. cit., p. 26.

[3] Op. cit.: "The evil fell mostly upon the poor."

[4] Op. cit., p. 70.

[5] L. c., p. 27: "In this epidemic also, experience showed that only the people of the lowest class were attacked."

[6] Op. cit., p. 53: "The poorer classes were the chief victims of the scourge."

[7] 'Del governo della peste, &c.,' lib. i, cap. 3, Bresc., 1721, p. 21.

[8] Wolmar, p. 31, Larrey, op. cit., i, 118, Delaporte, in Prus ('Rapport,' 329, 339), Delong, ib., 519, Masserano, ib., 514, Pruner, op. cit., 420, Aubert-Roche, 103, Clot-Bey, 190, 218, and others for Egypt; Russell, Lasperanza, in Prus ('Rapport,' 489), Delaporte and others for Syria; Thirk, l. c. 781, and Aubert for Asia Minor; Brayer, Cholet, in Prus ('Rapport,' 625), for Constantinople.

arrangements." In Dickson's account of the plague of 1876 in Irak-Arabi it is stated:[1] "The most palpable and evident of all the causes which predispose an individual to an attack of plague during an epidemic outbreak is *poverty*. No other malady shows the influence of this factor in so striking a degree; so much so, indeed, that Dr. Cabiadis styles the plague *miseriæ morbus*. In his experience (1876-77 in Bagdad) he found that the poor were seldom spared, the wealthy hardly ever attacked." This enables us, adds Dickson, to understand why Hillah, a filthy town crowded with a destitute proletariat, should have been decimated, and why Kerbela, one of the cleanest and most prosperous towns in Irak-Arabi, should have been almost entirely spared.

§ 128. Susceptibility of Different Races to the Morbid Poison.

In our critical survey it behoves us to glean the true lessons of experience as to differences in the amount of sickness and mortality among various *races and nationalities*. The observers of the last epidemics of plague in Egypt are almost absolutely unanimous in saying that the largest number of deaths from plague occurred among the negroes, next to them among the Berbers and Nubians, and in the third degree among the Arabs; while the Europeans were the most favorably treated, those from northern countries in the first place, and less so the southerners (Turks, Greeks, and Armenians). It appears to me to be doubtful whether we have here to deal with degrees of national predisposition. Probably it is the social element that gives the key to those differences; and that is the opinion also of Aubert-Roche and Cholet, who say that the nationalities who live amidst conditions of greatest wretchedness have suffered from plague most. This may serve also to explain the striking fact that plague on the slopes of the Himalaya has spread hitherto only among the natives, and has entirely spared the European officials, whose number, however, is small.

We must rate highly, therefore, the importance of this etiological factor for the development of plague; but we should not be justified in viewing the products of putrefaction as representing in themselves the *virus of plague;* and

[1] 'Papers,' p. 54.

the attempt in particular to refer the pathogenesis to the action of products of cadaveric decay, to the so-called *cadaveric poison*, must be held to have failed completely. This opinion, which had been entertained conjecturally before, has in recent times been placed on a seemingly firm basis, and invested with a halo of science, by Lagasquie[1] and Pariset,[2] the French commissioners sent to Egypt in 1828 to study the plague, and who regard Egypt as the proper seat and starting point of the disease. The same hypothesis has been adopted to explain the origin of the disease in the case of the recent outbreaks in Mesopotamia.

The observers in question consider it as proved (calling to witness Herodotus, Diodorus, and Strabo, who commend Egypt for its healthy climate) that plague had not occurred in Egypt previous to A.D. 543, but became epidemic there for the first time in that year. They point out that this first appearance of the plague falls exactly at the time when the embalming of bodies was given up, as a practice not consonant with the spirit of the Christian religion, and when burial was generally introduced. They go on to show that the sort of burial practised in Egypt has always been so very insufficient that the atmosphere all around has been filled with the products of cadaveric decomposition. "Thus it happened," say they, in closing their argument, "that with this most dangerous innovation a most dangerous disease was created. So long as corpses were embalmed there was no plague; when that practice ceased pestilence appeared. Is there a more conclusive and more notable piece of evidence in any science in which the facts are not ascertainable with mathematical certainty?"

In the groundwork of this argument, there is first of all the quite erroneous assumption that the disposal of the dead by embalming had been for thousands of years a practice among the whole population of Egypt.[3] But the hypothesis must lose all value whatsoever when we reflect that plague, on the testimony of Rufus, had been prevalent in Egypt in remote antiquity.[4] And the whole theory of plague originating

[1] 'Revue méd.,' 1829, Nov., 207.

[2] 'Memoires sur les causes de la peste,' Paris, 1837.

[3] Labat ('Annal. de la méd. physiol.,' 1834, xxv, 727) and Clot-Bey (op. cit., 192) have completely refuted this.

[4] Prus ('Rapport sur la peste'), who thinks it a "great and incontestable fact" that plague was unknown in Egypt at the time of the Pharaohs, and during the Persian and Macedonian empires, and under the Ptolemys, disposes of the inconvenient statements of Rufus with the words: "Everything leads us to believe that the cases observed there in those days must have been sporadic."

from the decomposition of corpses must fall to the ground, as the reflective physicians of the seventeenth and eighteenth centuries have pointed out,[1] when we recur to the history of wars in past times, and even in recent times. The populations of besieged towns have suffered for months together from destructive epidemics of typhus and dysentery; armies have been obliged to camp for long periods on battlefields, or close to them, when the air was charged with products of decomposition from the bodies of men and animals buried in heaps in shallow trenches; those scenes have been enacted over and over again, not only in the heart of Europe, but also near to, and even upon, the classical plague-ground of the East; and yet no outbreak of plague has been induced. One should not ignore the fact that an accumulation of the products of putrefaction taking place under such circumstances has not unfrequently encouraged the outbreak of the disease; but their action has been in no wise different from that of other products of decomposition. For the development of the disease and the formation of a plague-centre there is always required the access of the specific *virus of plague*. Those plague-centres extend just as far as the diffusion of the virus reaches to; but, where the virus comes not, no matter how unfavorable the hygienic conditions, there the immunity from the pestilence is complete.

§ 129. Native Habitats—Importation—New Foci—Disappearance from Egypt.

Connected with this discussion of the factors that favour the origin of plague, there is the question of the *native habitat of the plague-poison;* or, in other words, the question whether it is, or always has been, autochthonous wherever the disease has been prevalent or is prevalent still, or whether certain points within the plague-radius have been the original seats of the poison, and the occurrence of the

[1] Diemerbroeck, for instance, says (lib. i, cap. viii, Probl. iv, § 4): "Cadavera sive hominum, sive aliorum animalium putrescentia pestem non generare, docent multæ magnæ strages, in quibus talis cadaverum inhumatorum putrefactio nullas pestes induxit."

disease outside them has always been a consequence of importation of the poison.[1] As in the case of most of the infective diseases, so also for the plague, a definite answer to this question is beyond the bounds of possibility; and the obscurity which has at all times enveloped it has only been increased by the experience of the most recent outbreaks.

It is at least highly improbable that plague ever arose autochthonously on the soil of Europe. It is obviously impossible to follow now-a-days the track which the disease took in past centuries, and especially in the middle ages; but from the time that the attention of physicians and other authorities began to be directed to this important question, the conviction has continued to gain ground that the diffusion of plague over Europe had always to be referred to importation from the East.[2]

In the seventeenth and eighteenth centuries, when the outbreaks in Europe were rarer and within narrower limits, it was possible to follow these lines of diffusion with greater certainty; and for that period at least, as well as for the current century, the explanation which Eggerdes gave, in an excellent essay[3] on the plague, holds good:

[1] That plague counts among the communicable or contagious diseases, requires no further proof. Only an obscurantist adherence to preconceived opinions and vague notions can permit anyone nowadays to deny the communicability of plague, or to assert its autochthonous development from the "constitution of the air," or the "genius epidemicus," or from a transmutation of malarial fever or typhus. There may be differences of opinion as to the ways and conditions of its communication, but not as to the fact itself; and I believe I am justified, from the scientific point of view, in not discussing once more this threshed-out question, and in declining to add another to the legion of controversial articles on the contagiousness or non-contagiousness of the plague.

[2] Attention has justly been called to the great merits of many of the Italian physicians, such as De Bonagentibus and Massaria, in advocating the doctrine of contagiousness and in proving the importation of the disease into Europe from the East. But it may be inferred that German physicians also had at that period formed a correct opinion of the state of matters, from the accounts of the origin and progress of epidemics of plague in Europe which have been furnished by Agricola ('De peste libri iii,' Basil, 1554, 20), Cornarius ('De peste libri ii,' Basil, 1551-5) and Cirenberg ('Wider alle pestilenzische . . . . Fieber, ein gründtlicher Bericht,' Leipzig, 1564-7). The latter, speaking of the spread of the pestilence in 1549 from the East through Russia, Livonia, Lithuania, Prussia, Pomerania, and Silesia, says: "all of which, the people have brought *ex-contagio* from one country to another."

[3] 'Der grausamen Pest-seuche gründliche und wahrhafftige Abbildung,' &c. Bresl. and Leipz., 1720-28.

"Every medical man knows that contradictory or dissimilar opinions are held by those who write on the plague; but we shall not discover the true cause of this want of uniformity if it be not in the ignorance of those who leave such conflicting opinions behind them in their papers and books. One will have it that the plague comes out of the sky, another that it arises with bad and poisonous emanations out of the earth, and still others that plague is nothing else than a virulent fever, surpassing all other fevers in malignity. . . . Now, to get at the truth in these opinions, let us observe how the plague in 1679 was carried from Turkey to Hungary, from Hungary to Austria, from Austria to Bohemia, to Saxony, and to other parts of Germany. In like manner, that of 1705 was brought from Turkey to Podolia, from Podolia to Poland, and so on. . . . If my own history of plague and those of others are to be credited, this transference has taken place either through goods in which there had been concealed a certain material poisonous entity that could infect a man when he came in contact with it, or it had been handed on to others by men who were themselves already infected with it. Everyday experience has taught us that this has happened . . . without the sky or the stars, the earth or the air, or other diseases already prevailing, contributing in the smallest degree thereto. . . . If, then, the plague arises from such a material poison-entity, which can be carried *per fomitem*, as by a tinder, from one place to another, it follows necessarily that such poison must be organised, and that it can likewise multiply to infinity. . . . For, who does not see that from an infected thing first one man may catch, and from him another, and from the other a third, and so on until many hundreds, and even thousands, have caught the infection."

In these few artless words there is contained, in my opinion, more truth than in the many bulky volumes which contend for the autochthonous origin of plague on European soil not only in former centuries, but even in modern times and at the present day, basing their contention upon unproved assumptions and erroneously-interpreted facts—upon the assumption of a "typhous *genius epidemicus*," or such a "transformation of malignant fever into plague" as Eggerdes was adverting to.

It has been concluded, from the fact of plague breaking out at a time when typhus or malarial fever has been prevalent, that there is some nosological or etiological relationship between it and those diseases. There have been observations published, purporting to prove that there do occur transitions of typhus or malaria into plague, both for the epidemic as a whole and in individual cases; and it has been pretended that, in particular cases of typhus or typhoid, the symptoms have been aggravated by "plague-symptoms," that is to say, by inflammatory swellings of the lymphatic glands, and that malarial fever has been complicated

with symptoms of plague and has ultimately degenerated into a true epidemic of it. And while all these alleged observations have been brought forward as proofs of the autochthonous development of plague in Europe in recent times, it has been concluded further that the same must have taken place to a much greater extent in past centuries. The notion of a "typhous constitution of the air" may now be regarded as one that science has done away with; and, in further criticism of these arguments, it need only be said that the coincidence in time of two or more epidemic diseases does not by any means warrant us in at once concluding for their nosological and etiological identity or resemblance, that cases of typhus or malarial fever complicated with swollen lymphatic glands are far from constituting "cases of plague," and again, that in many of these alleged cases of "buboes" it has not been an affair of swollen lymphatic glands but of inflammation of the parotid, and that cases of that sort have never given rise to further "cases of plague" or to the development of a plague epidemic, not even under the worst hygienic conditions. Finally, as regards the alleged development of plague from malarial fever, we must set down the observations adduced in proof of it as errors of diagnosis. In numerous epidemics of plague, especially in recent times and at places where the disease had been rarely seen, and was therefore unfamiliar or quite unknown to the practitioners, these and other errors of diagnosis have played an unfortunate part; so much so, indeed, that often a long period has elapsed and the pestilence even reached its height before the faculty had arrived at a correct estimate of its character. It is certainly nothing to the credit of Seidlitz,[1] whose merits are generally so great, that he has given prominence to this error of "plague developing out of malarial fever," in his sketch of the epidemic of plague among the Russian troops in the Danubian Principalities in 1828-29; because two quite different diseases were prevalent together on the malarious soil of Wallachia, he has been led to promulgate an erroneous doctrine, which confronts us in the subsequent essay of Witt on the same epidemic (although it is here in a somewhat modified dress)[2] as well as in the accounts by Naranzi and by Beck[3] of

[1] He says in his report ('Abhandl. pract. Aerzte in Petersburg,' v, 213): "Thus the fever of the plague, transplanted from its native soil and divorced from the epidemic constitution favorable to it, changed into an intermittent. Why, then, should not intermittent fever, reversing the order, pass into bubo-plague, when circumstances and epidemic constitution are auspicious? Why should we not assume that an exalted ague-constitution may induce a disease, which is at other times sporadic and without any keenness of contagion, to take to itself an epidemic contagiousness?"

[2] He seeks to prove that the pestilence that occurred in the Russian army was not the plague, but a peculiar malady, arising from the endemic malarial fever of Wallachia raised to a higher potency and complicated with buboes, carbuncles, and the like, and called therefore the "Wallachian pestilence." Nothing more has been heard of this pestilence.

[3] 'Wien. med. Presse,' 1876, Nos. 23, 24. The plague in Bagdad he names "febris intermittens remittens que bubonica."

the epidemics of plague in Irak-Arabi. The latter, indeed, have been fairly disposed of by the publications of Colvill, Adler, Cabiadis,[1] and others; but it is usually a difficult matter to uproot an error resting on belief in authority, and it is therefore not surprising that in the last small epidemic of plague in 1878-79 in the Government of Astrakhan, where the diagnosis, to tell the truth, was made after the epidemic was over, the doctrine of the development of the pestilence from malarial fever should have found currency for a time. A greatly esteemed student of the medical history, who is an adherent of the doctrine of an autochthonous origin of plague on the soil of Europe, and is very sceptical as to the diffusion of the disease there solely by communication, remarks that "the conflict between the rigid ontological method and scientific pathology has never been brought out more sharply"[2] than in the discussions among the Russian military surgeons on the origin and character of the pestilence of 1828-29, one section of them holding to the opinion of Seidlitz, and the other taking the disease to be the true plague imported from Turkey, its place of origin. [Haeser adds that the controversy was settled in favour of the medical bureaucracy of St. Petersburg, and that "ontologismus" triumphed.] In my opinion that controversy arose because, on the one side, there were vague notions of "epidemic constitution of the air" and "transmutatio morborum" hindering a fair and intelligent view, while, on the other side, there was a firm adherence to the rational principle which Griesinger gives expression to when he says: "The plague is a perfectly specific disease and its causes must be specific." And this is a principle that must be maintained, if we are not to upset the whole nosology of the acute infective diseases, give up all progress in etiology, and end by allowing all the acute infections again to enter the category of "febris pestilens."

There is, in my opinion, not a single fact to show that plague ever had an autochthonous origin in Europe in the middle ages or subsequently. Since the seventeenth century, at least, every epidemic on European soil outside Turkey has owed its origin to importation of the morbid poison from the East, and that will probably hold good also for Turkey in Europe. The assertion of medical practitioners and other residents in Constantinople that an importation from Egypt and Syria has on every occasion preceded the outbreak of plague is by no mean conclusive; for many practitioners in Egypt, such as Wolmar and Bulard, think that it has always been brought to them from Turkey or Syria, while Robertson, Segur-Dupeyron,[3] and others, returning the

[1] Quoted by Dickson, in 'Papers,' p. 53.

[2] Haeser, 'Geschichte der Med. und der epid. Krankheiten,' iii, 671.

[3] In Prus, 'Rapport,' 592.

compliment, reject the endemic occurrence of plague in Syria, and locate the source of the disease in Egypt and Turkey—a style of investigation which leaves the inquirer as if suspended between heaven and earth. But it is worthy of note, in estimating the part that Turkey plays herein, that, on setting the plague-years in that country against those of Egypt and Syria, there are very few years when the plague has been in Constantinople without being at the same time in one or both of the other countries; and even these few exceptions might vanish if the history of pestilence in Egypt and Syria were better known to us than it is. Again, it is highly probable that the epidemics of plague which had occurred in Mesopotamia,[1] Persia,[2] Arabia,[3] the Barbary[4] States, and Morocco[5] up to the year 1839, or previous to the fresh outbreak in them, were not due to an autochthonous development of the morbid poison in those countries themselves, but to its importation from other regions. In favour of this view, there are the facts that plague has occurred very rarely in those countries, the various epidemics having been separated by intervals of ten or twenty or thirty years, and that the appearance of the disease has always coincided with its wide diffusion in neighbouring regions, from which an importation of it could, in many instances, be made out with certainty. Thus, of the large territory whereon plague has existed up to 1845, there remain only Egypt, Syria, and Asia Minor as the countries which one may venture to designate as the native habitat of the plague-poison; and the dictum of Rufus, "pestilentes dicuntur bubones, qui maxime circa Libyam et Ægyptum et Syriam observantur," appears to be justified for later times as well.

But this somewhat widely-accepted opinion has been a good deal shaken by facts observed within the last thirty years. In the first place we have become acquainted with the endemic occurrence of plague in the mountainous districts of Hindostan, where its beginnings, as Planck has

[1] Tholozan, 'Hist. de la peste en Mésopotamie,' 29.

[2] Id., 'Peste en Perse,' 41.

[3] Pasqua, in 'Papers,' 16.

[4] Lord, 'Algiers, with Notices of the Neighbouring States of Berbery,' London, 1835, ii, 170.

[5] Nion, quoted in Prus, 'Rapport,' 620.

shown, do not belong to the times when attention was first drawn to it, but to a much earlier period. Here we are dealing with an autochthonous centre of plague, which had perhaps been the source of the fourteenth-century epidemic known under the name of the Black Death; which has been probably connected with the outbreaks in various parts of Hindostan from 1815 down to recent times (see p. 510), and perhaps also with the epidemics in the Chinese province of Yun-nan; and, finally, which gives us a possible key to the latest outbreaks in Asia Minor. When the disease broke out in Arabia (Assir), Mesopotamia, and Persia after ten years had elapsed without so much as a trace of it appearing on the classical plague-soil of Egypt, Syria, and Asia Minor, the question naturally arose whence the plague-poison had come to those regions, which had been hitherto so rarely visited by it; and the repeated outbreaks there in subsequent times, while there has been still immunity in those countries that pass as the home of plague, have given rise to the suggestion that this may be a case of autochthonous origin, an opinion which Pasqua[1] inclines to as regards the disease in Assir, and Tholozan[2] as regards the epidemics in Mesopotamia and the outbreaks in various parts of Persia in 1876-77. This is certainly a very easy solution of the question; but it will hardly satisfy anyone who holds fast to those opinions, which we cannot well divest ourselves of, concerning the nature of the plague-poison and of the communicable morbid poisons in general. The undoubted power of reproduction which the poison possesses is enough to justify the assumption that it is an organic body, and the occurrence of the disease in regions where it is not indigenous leads us to suppose that this organic body, or its germs, have been brought thither from some place or other. Between the epidemics that have expired and the fresh outbreaks, we must suppose a continuity; and we shall perhaps not err if we seek for the means of this continuity, or for the intermediate links, in *sporadic cases* whose existence had been overlooked.[3] In his report on the epidemic of 1866-67 in Irak-

[1] 'Papers,' p. 16.

[2] 'Peste en Mésopotamie,' p. 90, and 'Compt. rend.,' 1877, vol. 85, 432.

[3] The controversy that was carried on so keenly in former times, as to whether

Arabi, Naranzi gives some short notes by Dr. Duthieul, of Bagdad, on the state of health during the years 1856—1867 in Bagdad, Kerbela, and other places in Mesopotamia; from these it appears that more or less numerous cases of "typhus or malarial fever with glandular swellings (buboes)" had occurred every year during that period, sometimes at one place, sometimes at another. The cases, he says, were not contagious, and they do not permit us to suppose, therefore, that we are dealing with cases of plague. The sporadic cases were followed by the epidemic outbreak of 1866, which Naranzi would also consider to be not the plague, but a "typhus loimodes non-contagiosus." I have already (p. 529) stated my view of what is to be made of these "bubonic malarial and typhus fevers;" here they were clearly sporadic cases of plague of a mild type, such as we now know from the observations of Cabiadis[1] and Tholozan[2] to occur elsewhere in Mesopotamia.

The state of health in all those countries where plague has broken out within the last thirty years is very imperfectly known to us, and probably also to the sanitary authorities of the governments in question.[3]

When plague-spots could exist for years, and perhaps for tens and hundreds of years, in the mountainous districts of Hindostan without the British Government knowing of them, we cannot summarily dismiss the suspicion that a similar state of things may have occurred in the hill-country of Assir, in the somewhat inaccessible mountains of Kurdistan, and in other localities, and that it had escaped the notice of the authorities, or had not been attended to by them, or had even in some cases been enveloped in secrecy and obscurity. One thing in proof of this is the fact that the first information of the sporadic cases of plague, which had existed

sporadic cases of plague occurred at all, has been settled definitely in the affirmative by the latest observations.

[1] 'Papers,' p. 51.

[2] 'Peste en Mésopotamie,' p. 73.

[3] To understand this, one has only to read Naranzi's account of the difficulties he met with among the crafty natives, in his inquiries into the pestilence in the Valley of Hindieh; to all his questions they returned ambiguous or evasive answers, and did all they could to conceal or distort the facts, so as to escape coercion of every kind, and particularly the hated sanitary regulations.

for many years in Mesopotamia, was obtained only after the disease had developed into a severe epidemic.

In further considering the origin of these latest epidemics in Nearer Asia, we have not only to think of the old plague centres in Egypt and Syria, but we should keep in mind also the possibility of an importation from India. Although the latter suggestion may seem at first sight unwarranted in consideration of the great stretches of country that separate India from Persia, yet it should not be forgotten that we know practically nothing of the state of health of those territories. Epidemics of plague may have occurred in them in connexion with the pestilence in India, which would not come to our knowledge for years (as in the case of the epidemic of 1871-73 in the south-west of China) and perhaps never. Moreover, there is known to exist a very significant transit of dead bodies between Mesopotamia and India, Persia, and Southern Turkestan ;[1] and there may have been some connexion, by means of imported morbid poison, between the repeated outbreaks in Mesopotamia and those in Persia.

I am well aware that this deduction leads me on to the slippery ground of hypothesis, and that many fundamental questions are left altogether unsettled by it. In particular, it gives us no help in our former difficulty concerning the circumstances, apart from the virus itself, that are needed to develop an epidemic out of sporadic cases of plague, or independently of such ; for it is self-evident that the influences which we have already considered, the states of weather and of social well-being, do not of themselves furnish the explanation. Finally, the hypothesis fails to explain the circumstances that have led to the *cessation*—I purposely avoid the word "extinction"—*of the plague in Egypt, Syria, and Asia Minor*. There is certainly much that is true, but

[1] Deutsch, in his account of the epidemic of plague in 1874 in Nedjef ('Wiener med. Blätter,' 1879, No. 11, 255), writes as follows: "As to the original cause, it is difficult to say anything; but filth and the transport of corpses from Persia, part of Southern Turkestan, India, and Beloochistan, may have had their share in giving rise to the disease. According to the statement of the custom-house at Nedjef, 600 corpses were brought into the town during the preceding month of May. At Kerbela, where I was garrisoned, 2000 corpses were brought into the town with great pomp and consigned to the catacombs where the saint Hussein lies buried."

there is not the whole truth, in the words which Aubert-Roche has chosen as a motto for his work on the plague—"La civilisation *seule* a détruit la peste en Europe, *seule* elle l'anéantira en Orient." Besides the system of quarantine rules, whose efficacy I cannot rate too highly for reasons that I have several times explained,[1] there is no doubt that the improvement in hygienic conditions which has been going on for centuries in the civilised States of Europe, has contributed very greatly to the gradual decrease and final extinction of the plague. But whether that is the whole solution of the problem is very doubtful. Still less can one admit this factor to be the sole explanation of the cessation of the pestilence in the East, although there also, and particularly in Egypt, the good effects of sanitary rules on the remission of the pestilence cannot be altogether ignored. These, and many similar questions in the history of national sickness, have no answer to get from science as far as we have gone; and it is well that we should recognise these limitations in our knowledge of the laws that regulate the appearance, the prevalence, the remission, and the extinction of pestilences.

§ 130. The Means of its Diffusion.

We are enabled to form an opinion as to the *means of spreading the disease*, with somewhat greater certainty than we can judge of its developmental conditions. It appears to be questionable whether the reproduction of the poison of plague takes place within the bodies of the sick, so that, on being eliminated therefrom and conveyed to other persons, it works as a disease-generator at once; whether plague, that is to say, should be reckoned with smallpox, typhus, and the like,

[1] See 'Vierteljahrschr. für öffentl. Gesundheitspfl.,' 1880, xii, 6. Tulloch, in his Report on the Health of Malta from 1837 to 1846 ('Statist. Reports on the Sickness and Mortality among the [British] Troops, &c.,' Lond., 1853, p. 97), says: "Considering the vast increase of intercourse between Malta and the Eastern shores of the Mediterranean, where this disease [plague] is so generally prevalent, and the reduction in the duration of the voyage by the employment of steam vessels, it is remarkable that this island should have entirely escaped during the ten years under review. There has been no increase, but rather a relaxation in the stringency of the quarantine regulations, so that the exemption cannot be attributed to any precaution of that nature."

as one of the contagious diseases proper. There is, at any rate, not a single fact in favour of this view that cannot be contested. Not to mention what I might almost call the ingenuous belief that the infection takes place only by way of *direct contact with the sick*, whereby we are not permitted to think of the poison entering the organism by any other channel than through the skin, we come to the experiences adduced in favour of contagiousness,—either specially devised experiments with inunction or inoculation of bubonic pus or with the blood of plague-patients, or the wearing of linen saturated with the perspiration of the sick, and such like. These possess no force whatever as evidence. On the one hand, the experiments[1] have been heedlessly planned, that is to say, they have been made under circumstances which did not exclude infection coming by other ways to the individual experimented on; and further, they have nearly always miscarried, so that, strictly weighed, there are more of them which tell against than in favour of the conveyance of the disease in this way, or, in other words, against the contagious character of plague. An interesting series of facts bearing on the question will be found in the communications of Cabiadis (published by Dickson[2]) on the mode of diffusion of the plague in the epidemic that came under his observation at Hillah and Bagdad in 1876-77. His conclusions are as follow: Mere contact with a plague patient is free from risk to the individual, provided that his stay in the atmosphere of the sick-room is not very long;[3] persons occupying the same dwelling with a plague patient, and avoiding all contact with him or his belongings from fear of infection, usually take the disease, whereas very few cases of sickness occur among those living in houses free from the plague, although they visit the sick and come boldly into contact with them, remaining, however, only a short time in the sick-room. All the physicians, surgeons, and assistants, who had paid visits every day to many hundreds of the

[1] A complete collection of them will be found in Prus, 'Rapport,' 79-85.

[2] 'Papers,' p. 51, and 'Brit. Med. Journ.,' 1879, March, 339.

[3] This reminds one of what used to be said by a French surgeon who contracted the plague in Egypt; he explained to his friends that they need have no hesitation in coming to see him as often as they wished, only they should not stay too long at his bedside.

plague-stricken in that epidemic, remained well, with the exception of one assistant, although the surgeons and the assistants, who opened abscesses and dressed and bound up wounds, were obliged to spend much time—more than the physicians—in the immediate proximity of the sick or in the closest contact with them. Cabiadis treated nearly 2000 patients without taking the slightest harm. Whether the plague-poison be eliminated or not from the sick, he declines to express an opinion; but he does not believe, at any rate, in the infecting property of pus from the buboes. On the other hand, there can be no doubt, in his opinion, that the air about the patient is one medium of conveying the disease; but clothes also, and linen and the like, which had been used by the patients, may serve to carry the morbid poison, as he had often had occasion to observe.

Neither these, nor any of the numerous other observations that have been published on the mode of conveyance of the disease, enable us to form a sound judgment on the part played by the patient in communicating the morbid poison —whether he multiplies it within his body or whether he merely becomes the carrier of the plague-poison which has been reproduced and become endowed with active properties outside him. The latter alternative I consider the more probable; and it is certain that healthy individuals may be the means of conveying it—a fact that was known and correctly appreciated by the physicians of the sixteenth century. "It may easily happen," says De Bonagentibus,[1] "that some persons who are insusceptible, unimpressionable, or slow to succumb, may preserve the seeds of plague in their clothes or even in the pores of their skin, and may infect the susceptible through carelessness or in ignorance."

But there can be no doubt of the diffusion of the morbid poison by goods; and this is another of the points on which there is incontrovertable evidence from the sixteenth and seventeeth centuries.[2] Proofs are given also by Kanold from the epidemic of 1709-10 in Prussia, by Antrechau from

[1] 'Decem problemata de peste,' Venet., 1556, p. 10.

[2] Forest ('Observat. et curat. med.,' lib. vi, obs. 22, Schol.) gives a case of communication of the pestilence by articles of clothing which a woman from Zeeland had brought to Alemaar; she distributed them among five children, who all died of plague.

the plague of 1720 in Toulon, by Desgenettes, Pugnet, and other French army surgeons, from the epidemics of 1798 and 1799 in Egypt and Syria, by Bulard from the epidemic of 1834-35 in Egypt, and by Ségur-Dupeyron[1] from observations made in the quarantine stations of Venice (1793 and 1818) and Syra (1832, 1834, 1837); and the last mentioned are so convincing that even the Paris Academy of Medicine, which maintained a very sceptical attitude towards the doctrine of the communicability of plague, could not but admit their importance.

§ 131. Nosological Characters—Pulmonary Hemorrhage—Identity of the Black Death with the Pali Plague.

Plague is an acute specific infective disease, characterised essentially by an affection of the lymphatic system, namely, inflammatory swellings of the external and internal lymphatic glands (buboes); to these are joined not unfrequently other local lesions, and a series of symptoms proceeding from general infection, which, however, are neither constant nor properly pathognomonic of the morbid process. In plague, as in all acute infective diseases, various gradations of form may be distinguished, according to the severity of the sickness: first an explosive type in which the patient dies of general poisoning within two or three days, without developing buboes to any considerable extent; next, severe or moderately severe cases with full development of the local process; and finally, a mild form in which there are buboes without any general symptoms, and the prognosis uniformly good. All those various degrees of development in the disease have been found side by side in all epidemics of plague; and the unity of the morbid process is further evidenced in the fact that all the affections, local or general, that complicate the process are met with more or less frequently in every epidemic.

Among the more frequent symptoms of a general kind are hæmorrhages, mostly in the form of petechiæ, but also hæmatemesis, epistaxis, and more rarely bleeding from the

[1] Prus, 'Rapport,' 600-602.

intestine, lungs, or kidneys. Unfortunately the available records of plague are poor in clinical facts; so that it is difficult to come to a definite opinion whether there are any considerable differences between the several epidemics as to the frequency of hæmorrhages in general, or as to the particular kinds of hæmorrhage. It is certain at least that bleeding from the lungs must be reckoned one of the rarest accidents in plague; in by far the larger number of all the inspections after death from plague, the lungs have been found to be normal.

In very many of the epidemics, bleeding from the lungs, as well as symptoms referable to the respiratary organs in general, have not been observed, or, at least, have not been mentioned; but the silence of observers on this point should not be taken to mean that they had overlooked the symptom, for many of them, in their elaborate descriptions of the group of symptoms, make mention in detail of other hæmorrhages, such as hæmatemesis, but make no mention of blood-spitting,[1] while others explicitly state that they had never seen bleeding from the lungs.[2]

[1] Pruner mentions (p. 395) the occurrence of hæmorrhages from the stomach, intestine, nose and kidneys, but he does not say a word about hæmoptysis. In like manner Clot-Bey (p. 36), who adds (p. 58): "Cough is a complication which has been mentioned by no one but Vinarius" (in his description of the Black Death). In the fifty-one clinical histories published *in extenso* by Aubert, hæmoptysis is not referred to once; in Fischer's report ('Jahrb. des ärztl. vereins zu München,' ii, 101), of the necropsies of those who died of plague at Cairo in 1835 it is stated: "The lungs were usually collapsed and empty of blood, a few cases having much half-coagulated black blood in their veins, but rarely distended with bloody serosity." Clot-Bey's record of upwards of 100 necropsies is to the same effect, only that an abundance of bloody mucus in the bronchi is spoken of as a somewhat frequent appearance. I have searched in vain for statements about blood-spitting in the reports of the plague in Malta and the Ionian Islands, particularly in the very exhaustive paper of Tully. Neither can I find any indications of severe lung affections in the medical reports of the plague in Prussia in 1709, published by Kanold, in the collection of papers on plague in Provence edited by Astruc, in the publications on the plague in Hamburg in 1710 by Diderich, and on that of 1708-13 in Austria by Werloschnig and Loigk, nor in the reports by Arnaud and Castaldi on the recent outbreaks in Benghazi and Mesopotamia.

[2] Russell, in his classical work on the plague in Syria (i, 97), says: "The breathing was natural in nearly all the cases. . . . Cough was not observed; the patients had no pain in drawing a deep breath." Again, at p. 105, he says: "I have had opportunities of seeing a flow of blood only from the nose and the womb. There have been, certainly, a few cases in which the blood came from the lungs; but, as the patients had been subject to spitting of blood before, I did not take these hæmorrhages as symptoms of plague, but rather as relapses of an old trouble." Robertson says nothing of blood-spitting in his account of the

There are a few epidemics in which mention is made of isolated cases of severe affection of the lungs, with or without hæmoptysis; for example, by Rivinus for the epidemic of 1680 in Leipzig,[1] by Lorinser for the epidemic of 1824-25 in Bessarabia,[2] and by Cabiadis for the epidemic of 1876-77 at Bagdad,[3] in which he had 1826 cases under observation, with 171 instances of hæmorrhage, but of these only six hæmoptysis. Finally, in a small minority of epidemics, pulmonary hæmorrhage seems to have been noticed more frequently; as, in the epidemic of 1636-37 at Nymwegen, according to Diemerbroeck,[4] in the Copenhagen epidemic of 1711, according to Bötticher,[5] and in the plague of Moscow in 1770 on the authority of Orraeus.[6]

In all these cases we have to deal with a more or less infrequent complication of plague with pulmonary hæmorrhage, a complication, however, which had hardly given a distinctive character to the epidemic. But this particular local affection presents itself in quite another light in the epidemic of the middle of the fourteenth century, which, under the name of the Black Death, came from the East and overran a large part of the Old World (see p. 497). It follows from the statements of all the chroniclers of this pestilence—medical and non-medical—that spitting of blood was one of the commonest phenomena in the course of the disease; and it appears from the account which Guido has left of the epidemic, that during the first two months of it at Avignon, when it was in its worst form, bleeding from the lungs furnished the *signum pathognomonicum*, while it was not until

plague in Syria. Chenot, in his work on the plague of 1755 in Wallachia (p. 86), says: "We have not noticed any spitting of blood." "Schlegel (in 'Hecker's Wissenschaftl. Annal. der Heilkd.,' 1831, xix, 156), basing on his observations in Bessarabia in 1819, remarks that plague differs from typhus in this, among other things, that it wants the pulmonary lesions which are so frequent in the latter.

[1] 'Diss. de peste,' Lips., 1681, cap. iii, § 17: "pessimum est, si per screatum aut tussim sanguinolenta, vel purulenta . . . . rejiciantur;" but at cap. v, § 37, he adds: "hæmorrhagia raro mihi obvenit," and in his summary of the symptoms he does not say a word about hæmoptysis.

[2] At p. 322 we find: "Many suffer from heavy breathing, and oppression and pain in the chest; violent coughing brings up tenacious mucus, and sometimes bright red frothy blood."

[3] Quoted in Dickson's report, 'Papers,' 51, and 'Brit. Med. Journ.,' l. c.

[4] 'De peste,' lib. i, cap. v, Schol.: "Tussicula, spirandi difficultas, sputum cruentum . . . . calamitosa erant," and, in lib. iii, cap. x: "Sputa cruenta quamvis in multis visa fuerint tamen valde communia non erant."

[5] 'Morbor. malignor. imprimis pestis . . . . explicatio,' Hamb., 1713-71.

[6] Op. cit., p. 112.

a later period that buboes were included therein. "The said mortality or plague," we read, "began with us in the month of January, and it lasted for seven months. It was after two different fashions; the first prevailed for two months, and was marked by continuous fever and spitting of blood, the patients dying within three days; the second type, which lasted for the rest of the time, had also continued fever, together with boils and sloughs in the external parts, chiefly in the armpits and groins, and death took place within five days." Although other writers do not divide the epidemic into two periods by so sharp a line, according to the type of the disease, yet they all emphasise the contrast between the simple bubonic disease and the form distinguished by blood-splitting, and they speak of the greater frequency of the latter.[1]

The history of plague furnishes but one analogous case to this peculiarly constituted type of the disease; we meet with it in the epidemics that were observed in Hindostan during the first forty years of this century.

Gilder, who treated plague-patients in the epidemic of 1820 at Ahmedabad, says, in his account (p. 195): "The natives distinguish two forms of the disease, one which they call *Ghant ka rog* or 'bubonic disease,' the other which they know by the name of *Kokla ka rog* or *Tuo ka rog*, meaning 'the cough.'" In the first form, the symptoms were: "General uneasiness of the frame, pains in the head, lumbar region, and joints; hard, knotty, and highly painful swellings of the inguinal or axillary glands, great thirst, delirium, &c.," or the recognised phenomena of ordinary bubo-plague. In the second form, there occurred "high fever, attended with burning pains about the scrobiculus cordis, skin intensely hot, . . . considerable pains in the chest and joints, delirium, great anxiety, the patient hawks up clots of blood, the difficulty of breathing increases, and death generally occurs on the second day of the attack." Whyte, who had the disease under observation in Kattiwar, says (p. 161): "The cases were here distinguished by fever and hæmorrhage, apparently from the lungs, without buboes; these were reported to be as surely and even more speedily fatal than those with fever and buboes;" and the statements of McAdam, Glen, and others are to the same effect. The same types occurred in the pestilence known by the name of Pali Plague from the circumstance of its breaking out first in the town of Pali in 1836. "The most fatal modification of the disease," says Forbes ('Bombay Transact.,' ii, 15), "from which no recovery has been known, sets in with . . . slight cough

[1] See my article, "Ueber die indische Pest," in 'Virchow's Archiv,' l. c.

and expectoration of blood; the cough appears to an observer more like a voluntary act to relieve oppression or constriction about the chest, than to be caused by pain or irritation. The body is covered with frequent clammy sweats, the countenance exceedingly anxious and wild, thirst urgent, the urine loaded with blood, which also oozes from the gums. The expectoration of blood becomes more copious . . . faintness and complete exhaustion come on, and a fatal syncope puts an end to the sufferings of the patient, generally within forty hours from the attack. . . . It is, however, by no means rare, to see the different forms mixed or merging in each other." Forbes adds to this sketch, the following remark having reference to the epidemics of 1815-20 in Cutch and Gujerat: "That the two diseases are the same, does not admit of a doubt, and it is also probable that this malady has, at intervals, prevailed epidemically throughout Marwer from a very remote period." The other authorities on this epidemic express a similar opinion; Keir ('Indian Journal of Med. Sc.,' 1841, ii, 247) specially mentions "fever with bloody expectoration but no swelling," and Maeclean (p. 22), referring to the same form of the disease, says: "In such cases buboes are not commonly observed, though they do occasionally co-exist with the inflammation [?] of the lungs."

Webb was the first to point out the resemblance between these epidemics of plague in India and the Black Death;[1] and I have myself endeavoured to carry out the idea further in a detailed historical account of "Indian Plagues." A consideration of the fact that all the testimony on the Black Death assigns its origin to the remote eastern parts of Asia, some assigning it, indeed, to India direct,[2] led me about the same time to throw out the suggestion "whether there was not more, perhaps, than a mere resemblance between the Black Death and these Indian plagues, whether we are not justified in surmising that a plague of that kind may not have arisen in India in the fourteenth century as well, and spread as a pandemic, under the name of the Black Death, from its place of origin over a large part of the inhabited

[1] Op. cit., p. xxiii: "The Pali plague exactly resembles the great plague, the Black Death."

[2] In the well-known poem of Fracastori, "De Syphilide," there is the following passage:

> "Bis centum fluxere anni, cum flammea Marte
> Lumina Saturno tristi immiscento, per omnes
> Aurorae populos, per quæ rigat æquora Ganges,
> Insolita exarsit febris, quæ pectore anhelo
> Sanguineum sputum exagitans—miserabile visu—
> Quarta luce frequens fato perdebat acerbo."

globe." Since the publication of that paper, nearly thirty years have elapsed, during which the subject has been often debated, having given rise to keen discussions more especially on the last outbreak of plague in the Government of Astrakhan. I may be permitted, therefore, to state in a few words the position that I hold at the present moment with reference to this question. How far my former conjecture is justified, I do not now inquire; but the resemblance of the Indian plague to the Black Death cannot very well be contested. I must remark, however, that it is an error to represent my view of the matter, as if I had assumed two different "forms" of plague, a Levantine and an Indian. I have expressly called the Indian plague only a "modification" of the bubo-plague, in the sense in which one speaks of hæmorrhagic malarial fever, the "fievre bilieuse hématurique,"[1] without implying therein more than a feature in the malarial process which gives a quite special character to certain epidemics and endemics of malaria, just as the pulmonary hæmorrhages do to the Black Death and to these Indian plagues. Further, I have expressly stated that the complication of bubo-plague with bleeding from the lungs has occurred in many other epidemics of plague, and that the Black Death and the Indian plagues differ from these only in so far as the complication, for reasons that we do not know, was much more frequent, amounting to a *signum pathognomonicum*, and accounting for the extraordinary malignancy of those epidemics. I have never asserted that this modification in the character of the disease is a property of *all* the epidemics that have occurred in India, to wit those that have been observed in Hindostan of recent years; nor do I believe it, inasmuch as there is no mention of blood-spitting in the accounts of the pestilence by Planck and Murray, which relate to the circumstances as they now exist.

In conclusion, there is one more objection to meet, which has been raised as to the character of the plague on the slopes of the Himalaya;[2] it has arisen out of the circum

[1] See p. 233.

[2] I do not think it necessary to reply to the objection that no "carbuncles, petechiæ, and vibices" have been noticed in the Indian plagues; I will merely call attention to the remark of Planck that petechiæ on the dark skins of the natives may easily escape the attention of the physician.

stance that the plague is endemic there at only a few points, that it does not become generally diffused, that it is limited to the natives (sparing the Europeans, who are English officials and few in number), that it appears, therefore, to deviate from one of the properties of bubo-plague, viz. communicability. In reply to this, it has to be said that the conclusion is an erroneous one, inasmuch as the communicability of the disease within its several centres has been ascertained by physicians, and is well known to the natives, who desert house and home, and even leave the locality. Further, it has been shown, as in the history of the disease in 1815—1838, that it has been by no means confined to the mountainous districts; and therefore there is a not very remote fear—as every one would admit—that it may again attain to a wider epidemic diffusion. Furthermore, the same phenomenon of the pestilence raging within narrowly circumscribed areas has been observed in the latest epidemics in Mesopotamia, Persia, and Benghazi (Tripoli), about whose character as plague there cannot be the slightest doubt. Finally, as regards the exemption of the European residents, that is explained according to the experience of all epidemics of plague, that the disease takes up its abode chiefly among the poorest classes living in filth and wretchedness, while it exempts, to a greater or less degree, those who are better off. This fact has been quite recently confirmed to the fullest extent by the above quoted observations of Dr. Cabiadis as to the absolute immunity enjoyed by the physicians in the epidemic of 1876-77 at Bagdad and Hillah.

CHAPTER XI.

TYPHUS.

(Typhus exanthematicus.)

§ 132. Historical: the Great Typhus Periods from 1708 to 1815.

The *history of typhus*[1] is written in those dark pages of the world's story which tell of the grievous visitations of mankind by war, famine, and misery of every kind. In every age, as far back as the historical inquirer can follow the disease at all, typhus is met with in association with the saddest misfortunes of the populace; and it is, therefore, a well-grounded surmise that the numerous pestilences of war and famine[2] in ancient times and in the Middle Ages, which

[1] I apply the word "typhus" in the sequel exclusively to petechial typhus. In the concluding chapter I use the word "typhoid" as a short and universally understood term, instead of the more cumbrous "abdominal typhus."

[2] I guard myself here in the most explicit manner against the assumption that I identify without further limitation the terms "war or famine sickness" with "epidemic typhus." The sickness of the populace, which breaks out in times of general misery, represents for the most part a mixture of various kinds of disease, such as diarrhœa, dysentery, scurvy, typhus, and frequently also malarial fever and typhoid. All these have not unfrequently been grouped together, both by chroniclers and historians, and even by medical authorities, as a single type of disease, which at the present day hardly admits of analysis, and in which only a few fundamental features of the various diseases can be distinguished. This aggregation is most marked in the descriptions, by chroniclers and historians, of the war and famine pestilences of antiquity; and it accounts for the vain endeavours of the historical critics, who regard the aggregate of symptoms as a single disease, to interpret them as corresponding to some one of the diseases now known to us, an endeavour which has sometimes led them to the not altogether sound conclusion that we have to deal with unknown and obsolete forms of sickness. The same misunderstanding meets us again, although in a lesser degree, in the accounts which the physicians of the seventeenth and eighteenth centuries have given of the then prevalent epidemics of "putrid fever," "mucous fever," and "bilious fever;" here again it is, in many instances,

are known to us, not from medical sources[1] but merely from the chronicles, had included typhus fever as a prominent figure among them.

The earliest references to typhus epidemics, having a degree of definiteness, date from the eleventh century. There is a notice of an epidemic in 1083 at the monastery of La Cava, overlooking the town of Cava near Salerno:[2] "In nostro monasterio in mense Augusto et Septembre crassavit pessima febris cum peticulis et parotibus . . . Hoc anno omnes fructus a vermibus consumti sunt." The same pestilence may possibly have been distributed over other parts of Italy; for we read in a chronicle of Brescia[3] for the same year: "Fames valida, grandisque mortalitas fere hanc civitatem delevit." In the chronicle of Bohemia[4] by Hagecius, mention is made, under the year 1095, of a swarm of grasshoppers which covered the whole country and laid waste the fields, and perished in heaps when the rains set in; the year after (1096) there was "an extraordinary mortality and infection among the people; no "plague-glands [buboes] were to be seen, but the complaint was of soreness in the head." Similar epidemics of "pain in the head" (or "head sickness," the term that afterwards came into general use for typhus) without "plague-glands" (thereby expressly distinguished from the plague) are often referred to in the German chronicles of the centuries following. Especially towards the end of the fifteenth century, typhus appears to have been widely spread in many countries of Europe; as in 1480 in Carinthia and Carniola (burning fever and pain in the head) in consequence of a famine;[5] in 1481-82 under the same circumstances in Friesland and other parts of Germany,[6] and in France;[7] in 1489 in Spain, during the campaign in Granada, having been imported, as was said,[8] by troops from Cyprus, where it was

plainly a question of the simultaneous occurrence of various forms of disease, which the most competent of critics are not always able to identify and distinguish.

[1] There are a few meagre references in the writings of the Greek and Arabian physicians to the occurrence of an eruption of small spots in the course of febrile disorders; but it is impossible to form any opinion, from the association of this symptom with other diseased appearances, whether it is the exanthem of typhus that is to be understood.

[2] In 'Chron. Cavense,' quoted by Corradi, 'Annali,' P. i, 101.

[3] Muratori, xiv, 873.

[4] In German by Sandel, 1596, vol. i, fol. 193.

[5] Valvassor, 'Topogr.-hist. Beschreibung des Herzogthums Krain,' Germ. ed. Laibach, 1689, lib. xv, 379: "A severe famine, and great mortality owing to the people quickly contracting a burning fever, which affected the head violently."

[6] 'Mansfeldische Chronik,' Frkft. a M., 1572, 402 b.

[7] Bouchet, 'Les Annales d'Aquitaine,' Poitiers, 1644, 287; Belcarius, 'Rer. gallic. comment.,' Lugd., 1625, lib. iv, cap. xvi, 96.

[8] Villalba, 'Epidemiologia Española,' Madrid, 1802, i, 112.

alleged to be endemic; in 1497 in Italy;[1] and in 1502—1504 in Germany, the disease being spoken of as "nova et dira aegritudo" (febres pestilentes, with pains in the head and hæmoptysis).[2]

It was at the commencement of the sixteenth century that the doctrine of typhus first began to be treated scientifically, particularly from the point of view of epidemiology; in the first instance by Italian physicians, basing on their observations made in the epidemics of typhus which were rife over a great part of Italy during the period from 1505 to 1530. Their publications were soon followed by numerous accounts of the disease from almost every part of Europe; so that, towards the end of the century, a rich epidemiological literature had been brought together, which affords us, along with the statements of the chronicles, some notion of the extensive area of typhus during that period. Still more did physicians give their attention and their literary skill to the severe and widespread epidemics of petechial fever which were fostered by the political and social confusions of the seventeenth and eighteenth centuries, and of the first thirty years of the nineteenth; the peoples of Europe suffered from repeated visitations during all those years, and the quantity of material that has come down to us for the use of the historian is indeed overwhelming. It would far exceed the limits that I have set before me in the execution of my task, if I were to give a complete enumeration of all the epidemics of typhus that are known to have occurred within that period, and there would be but little scientific interest in the constant repetition of facts of the same kind. I shall, therefore, confine myself to a chronological review of the more noteworthy episodes in the history of typhus; and that will serve at the same time as a survey of the area of diffusion of the disease in Europe during that period. I shall take occasion in the sequel to quote from epidemiological records such only of the particular details as are especially worthy of attention for the inquiry before us.

We have already seen that the first place in the pestilence-chronicles of the sixteenth century is occupied by the epidemic of typhus which

[1] See Corradi, 'Annali,' P. i, 357. Here again mention is made of a terrible famine.

[2] 'Chron. Hirsung.,' 596; Lichtenau, 'Chron. Argentor.,' 1609, p. 335; 'Mansfeldische Chronik,' 402 b.

spread in 1505—1508 over a large part of *Italy*,[1] where there was a severe famine. Fracastori, whose most valuable work "De morbis contagiosis"[2] contains the first description of typhus, based on his Verona experiences, mentions the epidemic under the name of "lenticulæ," "punctiula," or "peticulæ,"[3] as a contagious disease indigenous in Cyprus and adjoining islands, well known to the older physicians, but now observed for the first time in Italy. A second widespread epidemic of typhus in Italy—a true war-and-famine sickness—is met with in 1528—1530;[4] in connexion with which we have the outbreak of typhus in 1520 in *Germany*, whither the pestilence was imported from Italy.[5] There are other references to extensive Italian epidemics of typhus at Bologna[6] in 1540, Padua[7] in 1549, and Ancona[8] in 1552; and in Germany at Bamberg in 1540 (during war),[9] and in 1543. In *France* there is an account by Sander[10] of an epidemic of typhus in 1545 in Savoy and the adjoining French territory, which was known under the name (occurring often in later times), of *Trousse galante* "quod omnes juvenes et maxime vividiores e medio tollit." Coming to the second half of the sixteenth century, we meet with an epidemic of typhus in 1557, which extended over a large part of *France*[11] and *Spain*;[12] in the latter country, where it was known by the names of *Tabardillo*[13] and *Pintas*, it did not die out for many years ("quindecim fere annos"). Another severe epidemic began in 1566, as war-typhus, in Hungary—hence called "morbus Hungaricus—and spread over Austria, Bohemia, Germany, the Netherlands, and Italy; famine in many parts of Europe gave an impulse to it,[14] and it lasted until 1568. In 1572-74, the disease

[1] See Corradi, ii, 8.

[2] Cap. 6, Opp. Venet., 1584, 87. [His period was 1483—1553.]

[3] "Maculæ puncturis pulicum similes." The term "petechiæ" belongs to a somewhat later period, and is probably a corruption of "pestichiæ," diminutive of "pestis."

[4] Fracastori, l. c.; Massa, 'Lib. de febr. pestil., &c.,' Venet., 1556, 10, *seq.*, and 'Epist. med.,' tom. ii, cap. 9, Venet., 1558, 73; Salius Diversus, 'De febr. pestil. tract.,' Frcft., 1586, pp. 84, 93, *seq.*; Cardanus, 'Comment. in libr. Hippoc. de Victu,' Opp. x, 168; Mundella, 'Epist. med.,' Basil., 1543, 134; Montani, 'Opuscula,' ii, cap. 12.

[5] 'Mansfeld. Chron.,' 430 b.

[6] Susio, 'Libro del conoscere la pestilenca,' Mantov., 1575, cap. 9.

[7] Montanus, 'Consult. med. de febris cons.,' 43—49, 1572, p. 994.

[8] Amatus Lusitanus, 'Curat. med.,' cent. iii, cur. 71—74, 79—80, Venet., 1557, pp. 458, 467.

[9] Cornarius, 'De peste,' Basil, 1551, p. 8.

[10] In Forest, 'Observ. et cur. med.,' lib. vi, obs. 7.

[11] Coyttarus, 'De febre purpura epid.,' i, cap. 16, § 1578, 145.

[12] Mercado, 'De febris essentia,' lib. vii, Opp. Venet., 1611, ii, 599; Torœus, 'De febris epid. et novæ . . . natura,' Burgis, 1574, 26; Vallesius, 'Comment. in libros Hipp. de morb. popul.,' Colon., 1588, 815.

[13] "Tabardo" is the Spanish term for a cloak made out of coarse material, such as the country people wear.

[14] Jordanus, 'Pestis phænomena Tract.,' i, cap. 12, 19, Frcft., 1576, 120, 209, *seq.*

was prevalent, to a like extent,[1] in the Netherlands,[2] Germany,[3] France,[4] and Switzerland,[5] being again associated with war and failure of the crops. Among the isolated epidemics of typhus during that period, there deserve to be mentioned the notorious outbreaks on the occasion of the assizes held at Oxford[6] in 1577 (Black Assize) and at Exeter[7] in 1586. An area of severe famine-fevers, among which typhus was again conspicuous, was formed towards the end of the sixteenth century in *Lombardy*[8] (1578-88), *Italy* in general, with *Sicily*[9] (1590-92), a large part of *Germany*[10] (1591-92) and *Sweden*[11] (1597).

The seventeenth century saw an unusual number of pestilences, which spared no country of Europe, and in many regions committed terrible ravages. The development and extension of these scourges was for the most part connected, either directly or indirectly, with the turmoil of great wars, which arose now in one place, now in another, and shook the whole framework of European society to its foundations. Famine, due in part to the devastations of war, and in part to failure of crops from bad weather, was again a factor of no small importance in the general misery, and in the production of these disastrous pestilences, among which typhus played a leading part, along with plague, dysentry, and scurvy.

At the very beginning of the century, in 1604 and 1606, typhus had been prevalent in *Spain*, so widely in the latter year that it came to be

[1] Gemma, 'De naturæ divinis characterismis,' Antw., 1575, ii, 164, 217—259.

[2] Heurnius, 'Comment. in lib. II aphor. Hipp.,' lib. 1, § 23, Opp. Lugd., 1658, ii, 300; Gemma, l. c.

[3] Rhumelius, 'Histor. morbi . . . in Palatinatu super. Bavar., &c.,' Norbg., 1625, 83; Brunner, 'Bericht v. d. jetzt regierenden Haubtkraukheit,' &c., Lpz., 1580, and numerous notices in the chronicles.

[4] Ballonius, 'Epidemior. et ephemerid.,' lib. i, Opp. Genev., 1762, i, 25, 41.

[5] Plater, 'Observat. lib. ii,' Basil, 1680, 300.

[6] Stow, 'Annals,' Lond., 1615, p. 681.

[7] Hollingshed, 'Annals,' ii, 1547.

[8] Trevisi, 'De causis . . . febrium vulgo dictarum cum signis, sive petechiis, &c.,' Mediolani, 1588.

[9] Roboretus, 'De peticulari febre,' &c., Trident, 1592; Trunconius, 'De custodienda pueror. sanitate,' Florent., 1593, 251; Saxonia, 'De phœnigmorum . . . usu in febribus pestilentibus,' Patav., 1591, 2; Columba, 'De febri pestil. cognitione,' &c., Messan., 1596.

[10] Wittich, 'Bericht von dem . . . epidemialischen Fieber,' &c., Leipzig, 1592; Ruland, 'De morbo ungarico,' &c., Lips., 1610; Stetten's 'Chronik,' cap. ix, 726; 'Annales Gorlicenses,' in Hoffmann, 'Script. rer. Lusat.,' vol. i, part ii, p. 61.

[11] Ilmoni, 'Bidrag till Nordens sjukdoms historia,' ii, 125.

known among the people as "año de los tabardillos."[1] For the years 1607—1611 there are notices of typhus epidemics from various parts of Germany,[2] Italy,[3] and the north of Europe.[4] The widest diffusion of the disease was during the Thirty Years' War, in which the political and religious disagreements of Europe were fought out on German soil. *Germany*[5] herself suffered earliest and most severely, but the sickness afterwards reached *France*,[6] the southern provinces of which had already become the seat of widespread epidemics of typhus during the persecutions of the Calvinists and the conflicts connected therewith,[7] as well as the *Northern Kingdoms*, where the war between Sweden and Denmark and Norway furnished a new occasion for the extension of the fever.[8] Under the same circumstances typhus appeared in *England* during the Civil Wars,[9] and in the *Netherlands*,[10] while *Italy* was visited by extensive and severe epidemics especially in the famine years 1628-32 and 1646-50.[11] In the latter half of the century we find references to isolated but considerable outbreaks of typhus in 1649 and 1666 in many parts of *Prussia*,[12] in the latter year at Thorn,[13] whither the sickness was brought by the fugitives of the army of Carolus Gustavus of Sweden; further, in 1659-60 at many places in the north-west of Germany among the Swedish troops,[14] who carried back the disease to *Scandinavian* countries;[15] then on *French* soil—in Poitou in 1651,[16] and

[1] See Villalba, ii, 20, 28.

[2] Hoechstätter, 'Observ. med. Dec.,' i, § 5, Freft., 1674, i, 19; Libavius, in Hornung's 'Cista medica,' epist. 34, 51, 56, Norimb., 1625, 88, 160, 164.

[3] See Corradi, 'Annali,' iii, 15.

[4] Ilmoni, ii, 161, *seq.*

[5] Out of the large number of medical accounts relating hereto, I give references to the following as being the most noteworthy: Beckher, 'Fünfzen Fragen von der zehnjährigen Pest im Preussischen,' Königsb., 1630; Fonseca, 'De epid. febrili grassante, &c.,' Mechlin., 1623; Rhumelius, l. c.; Schmid, in Fabricius Hildanus, 'Obs. chir. Cent.,' vi, obs. 31, Opp. Freft., 1646, 534; Horst, 'Observ. med. Lib. de febr.,' i, obs. 33, Opp. Nürnbg., 1660, ii, 22; Hoechstätter, l. c., Dec. vii, 38; Lotich, 'Consil. et observ. med.,' lib. i, cap. vii, obs. 5—8, 15, Ulm, 1644, 86, 108; Neuerantz, 'De purpura liber.,' Lubec., 1648; Timæus von Güldenklee, 'Casuum medicin. lib. vii,' cas. 32, Opp. Lips., 1715, 345.

[6] Morellus, 'De febre purpur. epid. et pestil,' &c., Lugd., 1641.

[7] Riverius, 'Obs. med.,' cent. i, obs. 15, 43. Delph., 1651, p. 20, 30; Ader, 'De pestis cognitione,' &c., Tolos. 1628.

[8] See Ilmoni, l. c. ii, 191—194; Bartholin, 'Cist. med.,' Hafn., 1662, 444, 478.

[9] Willis, 'Lib. de febribus,' cap. xiv, Opp. Amstel., 1682, 113.

[10] Van Heers, 'Observ. oppido rarae in Spa et Leodii animadversae,' &c., Leod., 1645; Diemerbroeck, 'Observ. et curat. med.,' obs. 24—27 in Opp. Ultraj., 1685, 24.

[11] See Corradi, 'Annali,' iii, 56—138, 160—179.

[12] Keppler, 'Febris epid. Regiomont. anno 1649 . . . pernitiosa,' Elbing, 1650.

[13] Schultz, in 'Miscell. Acad. Leopold,' ann. iii, obs. 144.

[14] Esslinger, in Muralt's 'Schriften v. d. Wundarzney,' Berl., 1771, 591.

[15] Ilmoni, ii, 228.

[16] Pidoux, 'De febre purpurea,' &c. Aug. Pict., 1656.

in Burgundy,[1] in 1652 and 1666;[2] and finally in *Sicily*, where it was again associated with famine.[3] In *Germany*, typhus took a new start after the outbreak of the war of the Empire against France in 1673—1717,[4] as it did also in *Hungary* and *Austria* during the wars with the Turks in 1683—1718,[5] in *Sweden* and *Finland* as a disastrous famine pestilence in 1695—97,[6] and in 1688 in the *British Islands*, where the disease is said[7] to have begun in London in May, after which it spread over the whole of Great Britain, and began to spread over Ireland in July.

During the eighteenth century we find scarcely a year without references to epidemics of typhus, great or small, in one part of Europe or another; but there are four periods specially distinguished by the more general outbreak and prevalence of the sickness. The first of these joins on to the war pestilences which occurred, as we have seen, in Germany, Austria, and Hungary at the end of the previous century, and it extends to about 1720. Three severe epidemics in Ireland belong to this period, the first in 1708-10, the second in 1718-21, and the third in 1728-31, the two last having affected a large part of England and Scotland as well.[8] The second period includes the years 1734-44, in which typhus was prevalent over a large part of Eastern and Central Europe, mostly as a war pestilence in company with dysentery, malarial fever, and typhoid.

It appeared first among the *Polish* troops[9] in 1734 (during the War of Succession in Poland), and spread over *Silesia*,[2] *Prussia*, and the

[1] Marchant, 'Tract. de febre purpurata,' &c., Divione, 1668.

[2] Corradi, 'Annali,' iii, 249.

[3] Donckers, 'Idea febris petechialis,' &c., Lugd. Bat., 1686; Cardilucius, 'Tract. von der Pestilenz,' Nürnb., 1681; Scherf, 'De febre petech.,' &c., Argentor., 1676; Ruelius, 'De morbo epid.,' &c., Coburg, 1676; Lentilius, 'Miscell. med.-pract.,' Ulm, 1698, pp. 112, 122, 435-56.

[4] 'Fackh, 'Ephem. Acad. Leopold,' cent. v and vi, obs. 78; Löw, 'Acta Acad. Leopold.,' i, p. 23, ii, App. 25.

[5] Ilmoni, ii, 260—274.

[6] Wylde, 'Edin. Med. and Surg. Journ.,' 1845, April, p. 269.

[7] See Rogers, 'Essay on Epid. Diseases,' Dublin, 1734, 3-5; O'Connell, 'Morbor. acut. et chron. observ.,' Dubl., 1746, 65; Wintringham, 'Comment. nosologicum,' &c., Lond., 1733, 27—47; Huxham, 'Lib. de febribus,' cap. 8, Opp. Lips., 1784, ii, 94, and 'Observ. de aëre et morb. epid. ann. 1729,' ib. i, 58, *seq*; Strother, 'History of a Spotted Fever,' Lond., 1829; ref. in 'Edin. Med. Observ.,' i, 45—49.

[8] Lubienec, 'Hypomnemata morbi punctiularis,' &c., Posn., 1737; Camuset, 'Traité des fièvr. malignes épid.,' &c., Dresd., 1738; Meuder, in 'Select. med.,' Francofurt., i, 9.

[9] Meissner, 'Med. Silesiae. Satyræ,' vol. vi, 109.

shores of the *Baltic*.[1] About the same time it broke out in the French army and among the Imperial German troops on the *Rhine*,[2] and a few years later, after the breaking out of the Silesian war, in *Bohemia*,[3] whence it penetrated to Silesia. There is no doubt that many of the typhus epidemics which are reported about this time from the *Netherlands* and Central *Germany* were connected directly or indirectly with these foci of pestilence. During the same period (1740 and 1741) there were severe epidemics of famine fever in Ireland, Scotland, and England,[4] and in *Upper Italy*[5] in 1743-44.

In the third period, from 1757 to 1775, we find typhus springing either out of the turmoil of the Seven Years' War and the war between England and Spain, or out of the severe famine which visited a large part of Europe about the end of the third quarter of the century, and left a wide field for the disease to flourish in.

Commencing with *Germany* in 1757-61,[6] typhus was prevalent as a war-fever over a wide extent of country, and it is reasonable to suppose that the epidemics[7] observed in 1760 and 1761 in the eastern provinces of France (especially Burgundy) may have been connected with this, and may have been due to importation by French troops from the Rhine. There next grew up a severe war typhus in *Spain* in 1764, on the

[1] Wagner, 'Observ. de febre quadam acuta in tractu Germaniæ mari baltico,' &c., Lubec., 1737. (In 'Haller's Diss.,' pr. ii, 50.)

[2] Molitor, 'De febre continua maligna,' &c., Heidelb., 1736 (reprinted in Haller, v, 263); Kramer, 'Consil. med. de morbo castrensi,' &c., Aug. Vindel., 1735; Hoelder, 'Diss. de morbo castr. epid.,' &c., Jen., 1736; Vermalle, 'Disgression des maladies, qui regnent dans la plus part des villes de cette province,' &c., Spire, 1734.

[3] Brandhorst, 'Histor. febr. castr. petechialis epid.,' Lugd. Batav., 1746 (in Haller's Diss., pr. v, 421); Serinci, 'De febre maligna castrensi,' &c., Prag., 1743 (ib. v, 385).

[4] O'Connell, l. c., 325; Barker, 'Obs. on the Present Epid. Fever,' Lond., 1741, and 'Inquiry into the Nature . . . . of the Present Epid. Fever,' ib., 1742; Stark, 'Transact. of the Epidemiol. Soc.,' 1867, ii, 304.

[5] Corradi, 'Annali,' iv, 129.

[6] Baldinger, 'Von den Krankheiten einer Armee,' &c., Langensalza, 1744, 425; Monro (Donald), 'Account of Diseases most frequent in British Military Hospitals in Germany, 1761-63,' Lond., 1764, Germ. transl., Altenburg, 1766, pp. 1—48, 126; Lorentz, 'Morbi Gallor. castra trans Rhenum situ 1757—1762 infestantes,' Selestad, 1765, p. 145; Strack, 'Observ. med. de morbo c. petechiis,' &c., Caroliruh., 1796; Vogel, in 'Act. Moguntin.,' ii, 461; Ludwig, 'Adversar. med.-pract.,' Lips., 1769, i, 21, *seq.* (relates to Leipzig); Glaser, 'Verhalten bei der jetzo in Deutschland regierenden Flecktieber-Seuche,' Hildburgh., 1758; Kuhn, 'Nov. Act. Acad. Leop.,' tom. ii, obs. 71, 1761, p. 268; Grimm, ib., tom. iii, 1767, App. 143 (relates to Eisenach).

[7] Maret, in 'Mém. de l'Acad. de Dijon,' 1769, i, 125, and 'Mém. pour servir au traitement d'une fièvre épidémique,' Dijon, 1775.

entry into that country of the French army returning from Portugal; the sickness broke out first in Catalonia, spread to Estremadura and other provinces, and was in the end carried by the French troops into France. It was not completely extinguished in Catalonia until 1783.[1] In the form of famine fever, typhus showed itself during the first period in *Sicily* and over a large part of *Italy*[2] in 1764, and again in 1767, on the latter occasion confined mostly to Tuscany and the Romagna.[3] But it was in 1770-72 that it found its widest diffusion,[4] not only in the regions suffering under failure of crops and famine, such as Bohemia, Moravia, the eastern, northern, and central parts of *Germany*,[5] the *Netherlands*,[6] *Sweden*,[7] *Ireland*,[8] *England*,[9] and *Northern Italy*,[10] but in other places, such as the eastern and central cantons of *Switzerland*,[11] and the North of *France*,[12] whither it probably came by importation.

The fourth and by far the severest period of typhus in the eighteenth century occupies the last ten years of it; it begins with the revolutionary wars on French soil, and ends in the second decade of the present century, with the final retreat of the French army across the Rhine, the overthrow of the empire of Napoleon, and the restoration of peace.

[1] Masdevall, 'Bericht über die Epidemieen von faulen und bösartigen Fiebern.' From the Spanish, Braunschw., 1792; cf. Villalba, ii, 226, 236-37, 249, *seq.*

[2] Corradi, 'Annali,' tom. iv, 200—232.

[3] Ib., 242—267.

[4] See Hecker, 'Gesch. der neueren Heilkunde,' Berl., 1839, 150—200.

[5] Hecker gives a most complete synopsis of the titles of works, great and small, treating of the epidemic in those regions.

[6] De Man, 'Abhandl. von den bösartigen Faulfiebern,' &c. From the Dutch. Nymwegen, 1772 (reprinted in Collenbusch's 'Abhandl. holl. Aerzte,' i, Leipz., 1794); Von Elsaker, 'Spec. med. pract. febrem remitt. . . . Antverpiae et per plures Belgiae et Europae civitates . . . grassatam exhibens' (reprinted in Schlegel's 'Thesaurus,' ii, 315).

[7] Nils Apelbaum, in 'Läkare och Naturfosk.,' 1787, viii, 89. Cf. Ilmoni, iii, 365.

[8] Sims, 'Observ. on Epid. Disorders, with remarks on Fevers,' Lond., 1773. Germ. ed. Hamb., 1775, 93, 119, 150, &c.

[9] Grant, 'Enquiry into the Fevers most common in London,' Lond., 1771. Germ. ed. Leipz., 1775, 168—244, 283—306, 333—339.

[10] Corradi, 'Annali,' iv, 283.

[11] 'Anleitung gegen die Gefahr der Faulfieber,' &c., Zürich, 1782; see also Hecker, l. c., 186.

[12] Daunon, in 'Journ. de méd.,' 1773, xl, 24, for Boulogne; Bonté, in 'Hist. et mém. de la Soc. de méd.,' Année 1776, i, mém. 23, and Lepecq de la Cloture, 'Observ. sur les maladies epidem.,' Paris, 1776-8 (Germ. ed. Altenburg, 1788, 387), and 'Topogr. med. de la Normandie' (Germ. ed. Stendal, 1794, 420), for Normandy; Gaulmin des Granges, in 'Journ. de méd.,' 1772, xxxviii, 307, for Bourbonnais; De Villaine, ib., 1773, xl, 404, for Franche-Comté.

I must refrain[1] here from a detailed account of this period of pestilence, and confine myself to remarking that, as the oppression of war spared no country in Europe during those twenty years, so did the pestilences of war, and particularly typhus, following at the heels of the conquerors and the conquered, spread over all Europe and rise to a terrible height in those places where the visitation of war had been most severe. The medical authorities of those sad times have further admitted on all hands that typhus spread to other regions as well, which were directly out of reach of the military movements or the route of troops and beyond the range of sieges; that the conveyance of the disease through the peaceful intercourse between one place and another helped materially to spread it; and that it found in the universal distress caused by the war a very favorable soil wherein to flourish. The etiological importance of want is further shown very clearly in the recurrence of a severe epidemic of typhus in Ireland[2] in 1797 in consequence of failure of crops and famine; it lasted until 1802, and did not spare even Scotland[3] and England.[4] In these countries the effects of the war were not felt until later, and to a less degree than on the Continent.

§ 133. Recent Epidemics, Irish and Other.—Present Geographical Distribution.

Since 1815, when that period of typhus came to an end, the disease on European soil has only once, in 1846-47, attained the same general diffusion which the history of pestilence

[1] See the account in Haeser's 'Lehrbuch der Gesch. der Medicin,' 3, Aufl. iii, 531—537, and 595—618. Seitz ('Der Typhus, &c.,' Erlang., 1847, 124—213) gives a very full account of these typhus epidemics in Germany, especially in South Germany. For the epidemics of 1796 and following years in Italy see Corradi, 'Annali,' iv, 465, *seq.*

[2] Wylde, l. c.

[3] Stark, l. c.

[4] Currie, 'Med. Reports on the Effects of Water as a Remedy in Fever,' Lond., 1814, ii, 98—111; Willan, 'Reports on the Diseases in London, partic. from 1796 to 1800,' Lond., 1801, Germ. ed. Hamb., 1802, 138, 155; Murchison ('Continued Fevers of Great Britain') says that many hospitals for typhus had been founded in Great Britain chiefly in consequence of that epidemic.

presents to us so often in former centuries. Whole regions of the continent of Europe have remained almost entirely exempt from typhus since that time; the disease appears to be now confined to particular spots—we shall indicate them more particularly in the sequel—where it has the character of an endemic malady, and in which it develops to considerable epidemics under the influence of those factors that have always fostered the production of typhus; while these epidemics have not overstepped the limits of their indigenous foci to the same extent or with the same severity as they often did in former times. Even in the considerable epidemic of 1846-47, that peculiarity came out in a way not to be mistaken. The interest of this fact from the practical point of view makes it desirable to have a topographical survey of the typhus epidemics of the last seventy years; and I have endeavoured in the following pages to make as complete a collection as possible of the notices about them, and to sum up the results of my inquiries.

In no part of Europe does typhus bear the character of an endemic malady so decidedly as in *Ireland*. Even in the oldest chronicles of the country it is described as a permanent scourge of the people. Gerald Boate,[1] writing of the prevalent diseases, says: "Of this number is a certain sort of malignant fevers, vulgarly in Ireland called 'Irish agues,' because at all times they are so common in Ireland, as well among the inhabitants and the natives as among those who are newly come thither from other countries;" and all the later authorities, Harty, Barker and Cheyne, Graves,[2] and others, have written to the same effect. In the large towns it is never extinct,[3] and any considerable check to prosperity, most of all a bad harvest and famine, has nearly always been followed by the development of this disease into an epidemic.

Not to mention numerous lesser epidemics, typhus in Ireland has spread widely six times since 1815: in 1816-19 along with relapsing

[1] 'Natural History of Ireland,' Dubl., 1652.

[2] "It is a fact," says Graves ('Lond. Med. Gaz.,' 1837, Jan., xix, 571), "that typhus is more prevalent in this country than in any other European nation."

[3] Little, in the 'Dubl. Journ. of Med. Sc.,' 1835, March, p. 35; Popham (ib. 1853, May, 290) says: "It smoulders on until some spark kindles it into a flame."

fever, which furnished the largest contingent of cases;[1] in 1821-22, when it was the western or hilly counties that suffered;[2] in 1826-28 again universally, and in company with relapsing fever;[3] in 1836-37;[4] in 1846-47, the severest epidemic of this century in Ireland, the cases of sickness having been estimated at upwards of a million, or about one-seventh of the whole population;[5] and, lastly, in 1862-64, but only to a small extent.[6] Since the latter year there has been no considerable epidemic of typhus in Ireland.

The fact of close and active intercourse between Ireland and Great Britain, and most of all the emigration of the Irish, often on a large scale, to *Scotland* and *England*, will make it appear doubtful whether the somewhat frequent occurrence of typhus in the latter countries is to be ascribed to importation or to endemicity. In order to answer the question, we have, in the first place, to bear in mind that the severe epidemics of typhus which have visited Great Britain, have coincided with the epidemic diffusion of the disease in Ireland, and that the outbreaks in the latter have always preceded the rise of the epidemic in the former.

Thus, typhus appeared in Ireland towards the end of 1816, but in England[7] not until the spring of 1817, and in Scotland[8] not until the autumn. In 1821-22, when the epidemic affected only the West of

[1] See Rogan, 'Observ. on the Condition of the Middle and Lower Classes in the North of Ireland,' &c., Lond., 1819; Harty, 'Histor. Sketch of . . . . the Contagious Fever,' &c., Dubl., 1820; Cheyne, 'Dubl. Hosp. Rep.,' 1818, ii, 1; Barker and Cheyne, 'Account of the . . . . Fever lately Epidemic in Ireland,' 2 vols., Dubl., 1821. (The leading authority.)

[2] Account in 'Lond. Med. and Surg. Journ.,' new series, i, 399; Graves, in the 'Transact. of the Coll. of Physic. in Ireland,' 1824, iv, 316.

[3] Graves and Stokes, 'Clinical Reports,' pt. i, Dubl., 1827; O'Brien, in the 'Transact. of the Coll. of Physic. in Ireland,' 1824, v, 515.

[4] Graves, l. c., and in 'Dubl. Journ. of Med. Sc.,' 1839, Jan.; Babington, ib.; Kennedy, 'Med. Report of the Dubl. Fever Hospital,' Dubl., 1839.

[5] Accounts in 'Dubl. Journ. of Med. Sc.,' 1849, Feb., p. 64; May, p. 340; Aug., p. 1; Nov., p. 270; Donovan, 'Dubl. Med. Press,' 1848, Feb., p. 67; Kennedy, 'On the Connection between Fever and Famine in Ireland,' Dubl., 1847.

[6] Kennedy, in the 'Dubl. Journ.,' 1862, Aug.; Grimshaw, ib., 1866, May, p. 309.

[7] See Bateman, 'Account of the Contagious Fever,' &c., Lond., 1819 (relates to London); Prichard, 'Hist. of the Epid. Fever,' &c., Lond., 1820 (relates to Bristol); Edmonstone, in the 'Edin. Med. and Surg. Journ.,' 1818, Jan., p. 71 (relates to Newcastle); Hunter, ib., 1819, April, p. 244 (relates to Leeds).

[8] Duncan, 'Reports of the Practice in the . . . Royal Infirmary, 1817-18,' Edin., 1818.

Ireland, the sickness did not occur at all in England and Scotland. Again, in 1826-28, the diffusion of typhus was very slight in Scotland, and still less in England, and it broke out in both countries[1] much later than in Ireland; the same was the case in the epidemic of 1836-38.[2] The authorities[3] are nearly unanimous in ascribing the origin of the epidemic of 1846-47 in Scotland and England to importation from Ireland. Murchison's words are:[4] "There is abundant evidence to show that the fever was imported, to a great extent, by the Irish into the large towns of Scotland and England, and even to America. Apart from the circumstance that the epidemic commenced in Ireland, and first attacked those towns of Britain most accessible to Irish immigrants, it is well known that the Irish flocked over to Britain by the thousands, that in England and Scotland during the whole epidemic the majority of persons who suffered were Irish, and that at first they were almost exclusively Irish who had but recently left their own country. During the first three months of 1847, no fewer than 119,054 Irish immigrated into Liverpool alone; and so late as June, 1847, Dr. Duncan, the officer of health, stated that the fever was entirely confined to the Irish locality." The outbreak of typhus in England in 1856 was probably connected with importation by British troops returning from the Crimea;[5] while the epidemic that visited many parts of Scotland and England in 1862-64,[6] again coincided with a general diffusion of the disease in Ireland, although for this occasion there is no good evidence forthcoming of an importation of the fever.

The opinion that typhus in Britain results from continued importation from Ireland, receives further support from the fact that the localities in Scotland and England which are affected earliest, and most frequently if not ex-

[1] Stark, l. c.; Alison, 'Edin. Med. and Surg. Journ.,' 1827, Oct., p. 233; ib., 1828, Oct., p. 405; Burne, 'Treatise on Typhous Fever,' Lond., 1828.

[2] Craigie, 'Edin. Med. and Surg. Journ.,' 1837, April, 285; Henderson, ib., 1839, Oct., 429; Anderson, 'Edin. Monthly Journ.,' 1842, Oct., 936; Cowan, 'Vital Statistics of Glasgow,' 1838; West, 'Edin. Med. and Surg. Journ.,' 1838, July, 118; Roupell, 'Treatise on Typhous Fever,' Lond., 1839.

[3] Orr, 'Edin. Med. and Surg. Journ.,' 1848, April, 363; Steele, ib., July, 145; Paterson, ib., Oct., 371; Stark, ib., 1849, April, 380; Bennett, 'Edin. Monthly Journ.,' 1847, Oct., 299; Robertson, ib., 1848, Dec., 368; Hughes, 'Lond. Med. Gaz.,' 1847, Nov., 923, 970; Laycock, ib., 787; Bottomley, 'Prov. Med. and Surg. Journ.,' 1847, Dec., 701; Gairdner, 'Edin. Med. Journ.,' 1859, July, p. 45.

[4] L. c., 48-9.

[5] Murchison, p. 51.

[6] Buchanan, 'Transact. of the Epidem. Soc.,' 1865, ii, 17; notice in the 'Lancet,' 1864, Dec.; Hamilton, ib., 1867, Nov., 608; Martin, 'Brit. Med. Journ.,' 1863, July; Thompson, 'St. George's Hosp. Rep.,' 1866, i, 47; Allbutt, ib., 61; Davies, 'Med. Times and Gaz.,' 1867, Oct., 427; Russell, 'Glasg. Med. Journ.,' 1864, July; Beveridge, 'Lancet,' 1868, May, 630.

clusively, are precisely those which the Irish immigration touches first and foremost; that the disease is found chiefly in the great centres of industry and commerce; and that those counties which are withdrawn from the stir of traffic and the Irish immigration, and are given up to agriculture and cattle rearing, have been visited by typhus only on rare occasions and under exceptional circumstances. The opinion of Graves,[1] one of the best observers of typhus epidemics in Britain, is that, "when typhus occurs in Scotch or English towns, it is always in consequence of importation of the disease from Ireland;" and that opinion has been materially confirmed by the observations made in more recent times.

Russia forms a second great endemic centre of typhus on European soil.

The medical and topographical data from this great group of countries are too scanty to permit of our laying down accurately the limits of diffusion of the disease as an endemic; definite evidence comes only from Poland, the Baltic Provinces,[2] St. Petersburg,[3] the Government of Viatka and adjoining districts on the Volga,[4] the Caucasus,[5] Siberia, and Kamschatka.[7] Of the great typhus epidemics of recent date in Russia, about which we have information from physicians, the following deserve special mention:—The famine fever of 1821-22 in the Government of Novgorod;[7] the epidemic of 1830-31 in Poland and Courland, where the disease was brought by troops;[8] the general diffusion of typhus in 1842-46 throughout Poland,[9] Courland, Esthonia, Livonia, Kowno,[10] Wilna, Moscow,[11] Novgorod,[12] and other governments; the epidemic of typhus during the Crimean War (1854-56),[13] followed by

[1] 'Lond. Med. Gaz.,' 1837, Jan., p. 57.

[2] Œsterlen, 'Deutsche Klin.,' 1852, No. 36; Uhle, 'De typho in regionibus septentrionalibus observato,' Jen., 1860; Bohse, 'Dorpater med. Zeitschr.,' 1874, v. 1.

[3] Herrmann, 'Petersb. med. Zeitschr.,' 1870, 385.

[4] Jonin, 'Med. Ztg. Russland's,' 1849, No. 45.

[5] Liebau, 'Petersb. med. Zeitschr.,' 1866, 289.

[6] Bogonodski, 'Med. Ztg. Russ.,' 1854, 9.

[7] Auer, 'De typho contagioso, &c.,' Dorp., 1825.

[8] Bidder, in the 'Hamb. Magaz. der Hlkd.,' xxvi, 1.

[9] Notice in the 'Med. Ztg. Russ.,' 1847, 86.

[10] Notice in the 'Neue med.-chir. Ztg.,' 1846, iii, 252; Stäger, 'Rigaer Beitr. zur Heilkd.,' ii, 299.

[11] Heimann, in 'Casper's Wochenchr. der Heilkd.,' 1847, 733.

[12] Bardowsky, 'Med. Ztg. Russ.,' 1850, 172.

[13] Garreau, 'Gaz. méd. de Paris,' 1855, Nos. 43, 44, 1856, Nos. 6, 7, 14; Jacquot, 'Du typhus de l'armée d'Orient,' Paris, 1858; Baudens, 'Gaz. des hôpit.,' 1856, No. 68; Alferijef, 'Med. Ztg. Russ.,' 1858, No. 31.

the spread of the disease over a large part of Southern Russia;[1] the epidemic of 1866-68 in the Baltic Provinces, St. Petersburg, and other places in the north of the Empire;[2] the epidemic of 1874-76 in St. Petersburg,[3] Warsaw, Minsk, Moscow, Novgorod, Jaroslav, Kherson, Pensa, and Siberia;[4] and finally, the disastrous war-pestilence of the Russo-Turkish War (1877-78), in which the Army of the Caucasus was decimated by typhus.

In Germany, from the extinction of the severe war-pestilence of 1813-14 down to 1847, typhus showed itself only in isolated epidemics. It was chiefly *Upper Silesia*[5] and the districts of *West Prussia*,[6] occupied by a Slavonic stock, that formed the seat of these epidemics; they fall between 1831 and 1840, and they furnish evidence that the disease was endemic in those districts.

In the report from Upper Silesia for 1833, we read: "Although there can be no doubt that typhus has sometimes been introduced by means of infection from Poland and Galicia, yet it is not less certain that in the east and south-east of the department, it has more often arisen of itself, or, been generated *de novo*. The Sclavonic nationality, the customs of the inhabitants, and the great want and penury in which they live, particularly the want of wholesome food, appear to have been especially favorable to the development of the disease." Neumann also states,[7] in his sanitary report for the Circle of Strasburg (Department of Marienwerder), that he had seen the disease epidemic every year in one village or another.

There appear also to have been epidemic centres of typhus during that period in *Westphalia*[8] and the *Rhine*[9] country; at

[1] Account, ib., 1858, 59; Koch, ib., 1857, No. 1.

[2] Bohse, Herrmann, l. c.

[3] Herrmann, 'Petersb. med. Woch.,' 1876, Nos. 16, 17.

[4] Ucke, 'Vierteljahrschr. für gerichtl. Med.,' 1879, Oct., 344.

[5] 'Sanitätsberichte von Schlesien' for the years 1831 (p. 46), 1832 (p. 70), 1833, ii, 63.

[6] 'Sanitätsberichte des Königsb. Med. Coll.,' 1836, ii, 12, 1837, i, 14.

[7] 'Rust's Magaz. für die ges Heilkd.,' 1840, lvi, 177.

[8] Vogtt, 'Versuch einer geschichtl. Darstellung des ansteckenden Typhus,' Coesfeld, 1828; Seiler, in 'Hufeland's Journ.,' lv, pt. 2, p. 34; Rüther, in 'Casper's Wochenschr.,' 1840, 797, and in the 'General-Sanitäts-Bericht von Westfalen,' for 1838, p. 42, 51, for 1840, p. 21, for 1841, p. 26, 39, 77, for 1842, p. 19, 34, for 1843, p. 35, for 1844, p. 32, for 1845, p. 30.

[9] Brockmüller, in 'Horn's Arch. für med. Erfahr.,' 1822, ii, 320; 'General-Ber. des Rhein. Med. Colleg.,' 1828, p. 14, 1832, p. 25, 1833, p. 28, 1834, pp. 16, 20, 23, 1836, p. 21, 1837, pp. 14, 16, 1838, pp. 14, 18, 1840, p. 15, 1841, p. 18, 1842, pp. 18, 19, 1843, p. 11, and 1845, pp. 8, 10.

least, the unusual frequency of the disease at many points within these provinces may be so interpreted.

In Berlin we meet with typhus in 1828 and 1837, in isolated cases and to a slight extent, having been clearly imported both times from without, and on the latter occasion confined to the so-called tenement houses filled to over-crowding by the proletariat;[1] in 1824 at Marburg, where the epidemic began in an orphanage;[2] in 1832 and 1837 at Prenzlau;[3] in 1831 1834, and 1844 at Halle, but only in the filthiest part of the town and amongst the poor;[4] in 1838 at Goslar[5] and Meldorf (South Dittmarschen);[6] and finally, as gaol fever in 1828 and 1838 at Posen,[7] and in 1831 at Hamburg, where the disease sprang up in a house of detention for tramps, which was overcrowded during the cholera time.[8]

In 1847-48 typhus came to a considerable height in *Upper Silesia* where the harvest had been a failure and want was severely felt; but it did not extend to other districts of the province.[9] It is impossible to decide whether the epidemics of 1848 in Halle[10] and Minden (Hanover)[11] were connected with this pestilence in Upper Silesia. We find a more extensive epidemic of typhus in 1853-56 in the *Palatinate*,[12] which had been quite free from it since 1814; and in the same period (1855 and 1856) a new and more severe outbreak occurred in Upper Silesia,[13] which extended this time to Lower Silesia and produced a considerable epidemic in Breslau.[14] Further, there was a considerable outbreak in

[1] Bartels, 'Die gesammten nervösen Fieber,' Berl., 1838, ii, 378.

[2] Rothamel, in 'Horn's Archiv für med. Erfahr.,' 1827, ii, 938.

[3] 'Sanitäts-Bericht für die Prov. Brandenburg,' 1833, p. 99, 1837, p. 30.

[4] Staberoh, 'Diss. de typho exanthem. Halis epid.,' Hal. 1834; Bertog, 'De typho Halis vere anni 1844 observ.,' Hal., 1844.

[5] Forcke, 'Hannov. Annal. für Hlkd.,' v, 306.

[6] Michaelsen, in 'Pfaff's Mitth. a. d. Gebiete der Med.,' 1841, pt. 5—6, p. 1.

[7] Herzog, in 'Rust's Magaz.,' xxxvi, 432; Marcinkowsky, 'Preuss. med. Vereins-Ztg.,' 1834, No. 11; Cohen, in 'Rust's Mag.,' lvi, 213.

[8] Schmidt, 'Hamb. Mittheil. a. d. Geb. der ges. Heilkd.,' ii, 243.

[9] See particularly Virchow in 'Virchow's Archiv,' ii, 1; Dümmler, ib., 323; Deutsch, in 'Preuss. med. Vereins-Ztg.,' 1849, Nos. 33—35; notice in 'Casper's Wochenschr. für die ges. Hlkde.,' 1849, 577; Bärensprung, in 'Häser's Archiv für die ges. Med.,' x, 448.

[10] Lodderstädt, 'De typho Halis observ.,' Hal., 1849.

[11] Luchmund, 'Hannov. med. Corrspdzbl.,' 1850, i, No. 8.

[12] Accounts in the 'Bayr. ärztl. Intelligenzbl.,' 1854, p. 435, 1856, p. 503.

[13] Frank, in 'Virchow's Arch.,' x, 411; Rosenthal, ib., 512.

[14] Ebers, 'Zeitschr. für klin. Med.,' ix, 39; Grätzer, 'Statist. der Epidemie von Typh. exanthem. in Breslau,' Bresl., 1870; v. Pastau, 'Die Petechialtyphus-Epidemie in Breslau, 1868 bis 1869,' Bresl., 1871.

1855 at many places in the Odenwald,[1] where there had been a famine the year before, and also in Neckargemünd,[2] and in the neighbourhood of Göttingen.[3] From 1857 to 1866 typhus showed itself in Germany only in isolated and for the most part closely circumscribed epidemics; but since 1867 that country has been twice visited by severe outbreaks of the sickness. The first of these, in *East Prussia*, developed in 1867, at a time of bad crops and famine, among railway navvies who were living in frightful misery; it appeared first in the circle of Lötzen, and spread thence very extensively over the circles of Stallupöhnen, Pilkallen, Ragnit, Insterburg, and Tilsit, and did not die out altogether until the winter of 1868-69.[4] Probably connected with this epidemic, was the outbreak of 1868 in a few districts of *West Prussia*,[5] perhaps also the epidemic of 1868-69 in Breslau, where relapsing fever had been prevalent the year before,[6] and finally the appearance of the disease in Berlin, where it has never since been altogether absent, prolonging its existence in the lowest kind of lodging-houses and taverns (the so-called "Pennen"), and being fed by new importations of the morbid poison.[7] The cases in Berlin were especially numerous in 1873[8] and 1878-79,[9] but exclusively among the poor. Under the same circumstances as in East Prussia, but quite independently of the epidemic there, typhus broke out in the circle of Franzensburg (Department of Stralsund) among the men working on the roads, but it was confined within narrow limits.[10] It is diffi-

[1] 'Mittheilungen des bad. ärztl. Vereins,' 1856, No. 15, 113.

[2] Guerdan, ib., No. 22, 169.

[3] Theuerkauf, in 'Virchow's Archiv,' xliii, 35.

[4] Naunyn, 'Berl. klin. Woch.,' 1868, 237; Haffner, ib., 412; Becher, ib., 502; Müller, 'Die Typhus-Epid. vom Jahre 1868 im kreise Lötzen,' Berl., 1869; Passauer, 'Ueber den exanthem. Typhus,' &c., Erlang., 1869.

[5] Zülchaur, 'Berl. klin. Woch.,' 1868, 135.

[6] Graetzer, v. Pastau, l. c.

[7] Fraentzel, in 'Charité-Annal.,' 1876, i, 339.

[8] Zülzer, 'Viertelj. für gerichtl. Med.,' 1874, Jan., 182; Obermeier, 'Berl. kl. Wochenschr.,' 1873, Nos. 30—31.

[9] Salomon, 'Bericht über die Berliner Flecktyphus-Epidemie im Jahre 1879,' Berl., 1880 (reprinted in the 'Archiv. für klin. med.,' 1880, xxvii, 456).

[10] Treskow, 'Berl. klin. Woch.,' 1868, 72, and 'Beob. über die Wirkung des kalten Wassers im Typhus exanth.,' Greifsw., 1868.

cult from the facts before us to say whether we should connect with that epidemic the sporadic cases of typhus observed at Stettin[1] in 1868 and at Greifswald[2] (along with relapsing fever) in 1867-68. One of the most interesting occurrences during this period was the introduction of typhus in 1867 and 1868 from the eastern centres of infection by Slavonian hawkers (selling mouse-traps) into a large part of Central and Western Germany, particularly into many places in Thuringia,[3] into Driebrg and Münster,[4] Marburg,[5] Frankfort-on-Main,[6] Mayence[7] and other places, in none of which did it come to be widely diffused. The second severe epidemic of typhus of recent years in Germany was again in Upper Silesia, in 1876 and 1877. As early as 1874 and 1875 sporadic cases had been seen at many points, and in the summer of 1876, the disease assumed the character of an epidemic and continued as such until the end of 1877; the circles of Beuthen, Zabrze, Kattowitz, and Pless suffered most, Oppeln to a less extent, while Lower Silesia escaped altogether.[8]

The amount of typhus, during the last sixty years, in the States of the *Austrian Empire* is very imperfectly shown in the accounts that are available. Its comparative frequency in *Galicia*,[9] where typhus was general in 1846-47, as it was also in Austrian Silesia,[10] points to endemic foci of it there; and that may apply also to certain districts of *Bohemia*,[11]

[1] Wegener, 'Jahrb. für Kinderhlkde.,' 1868, i, 35.

[2] Mosler, 'Erfahr. über die Behandl. des Typh. exanth., &c.,' Greifsw., 1868.

[3] Seidel, 'Deutsche Klinik,' 1868, 426.

[4] Kaiser, 'Corrspdzbl. für die mittelrhein. Aerzte,' 1868, 11.

[5] Schnegelsberg, 'Ueber das Vorkommen des Typhus in Marburg, &c.,' Marb., 1872; Hartwig, 'Ueber Typh. exanth. in Ockershausen,' Marb., 1868.

[6] Varrentrapp, 'Corrspdzbl. für die mittelrhein. Aerzte,' 1868, 145.

[7] Masserell, ib., 140.

[8] Riehter, 'Berl. klin. Woch.,' 1877, No. 17; Schlokow, 'Deutsche med. Woch.,' 1877, No. 14 ff; Pistor, 'Vierteljahr. für gerichtl. Med.,' 1878, July, 69.

[9] See Mauthner, in 'Hufeland's Journ.,' 1834, lxxviii, pt. 4, p. 46; notices in 'Oesterr. med. Jahrb.,' n. s., i, 63, 67; Rohrer, ib., 1845, iii, 353; Maysel, ib., 1841, März, p. 267; Leiblinger, 'Wiener med. Woch.,' 1868, Nos. 54, 55.

[10] Suchanek, 'Prager Viertelj. für Hlkde,' 1849, i, 107.

[11] Popper ('Zeitschr. für Epidemiol.,' 1876, ii, 290) instances five severe epidemics in Prague in the period from 1820 to 1867, viz. in 1825, 1836, 1847, 1855, and 1867.

where the disease was general in 1847-50.[1] In *Lower Austria*, apart from a few small local epidemics at various parts of the Arch-Duchy, we have accounts of typhus only from Vienna, where there were severe epidemics in 1842,[2] 1853, 1855-56,[3] 1858-59,[4] 1862-63,[5] 1870-71,[6] and 1875.[7] The information as to typhus in the other possessions of the Austrian crown, including *Hungary*, are so extremely scanty that it is quite impossible to form any opinion as to the manner and frequency of its occurrence therein, and that is the case also for the *Danubian Principalities*,[8] for *Turkey*,[9] and for *Greece*. It would be interesting to find out whether the statement of Fracastori, that typhus had spread to Western Europe from Cyprus and islands adjoining, might apply at the present day, or, in other words, whether the disease is still endemic in the *Islands of the Mediterranean*. I can learn no more than the fact, taken from the English Army Medical Reports for 1817-46, that out of 111,774 cases of illness in the Ionian Islands during those thirty years only 10 were cases of typhus, and that 59 cases of typhus were observed in a total of 70,351 admissions in Malta. It remains doubtful whether the severe epidemic of "continued fever" in Malta in 1865 was typhus fever.

Among the European foci of typhus, *Italy* ranks next to Ireland and the countries with a Slavonic population. I take the following data, relating to typhus in that country from

[1] See Schütz, in 'Prager Viertelj. für Med.,' 1849, ii, 34; Finger, ib., iii, 5. According to the account by Mezler (ib., 1850, iv, Supplement, 15), there were 11,180 cases of typhus from Nov., 1849, to May, 1850, among 585,000 effective troops quartered in Bohemia.

[2] Dietl, 'Oesterr. med. Jahrb., 1843, Jan., p. 33.

[3] Mayr, 'Wochenbl. der Zeitschr. der Wiener Aerzte,' 1856, No. 47.

[4] Nader, 'Zeitschr. der Wiener Aerzte,' 1859, 417.

[5] Haller, 'Oest. med. Jahrb.,' 1863, pt. 4.

[6] Breslauer, ib., 1871, 523.

[7] Oser, ib., 1877, 425.

[8] In Wallachia, according to Barasch ('Wien. med. Wochbl.,' 1854, No. 26), typhus has been more frequent since 1849; there were severe epidemics there in 1853 and 1877.

[9] There were severe epidemics during the wars of 1854-55 (see under Russia, p. 559), and 1878-79. Marroni says ('Arch. de méd. nav.,' 1868, July, 44) that typhus occurs in the central prison of Constantinople every year in autumn and winter.

1816 to 1850, from Corradi's very thorough investigation into Italian pestilences, published in his 'Annali delle Epidemie occorsi in Italia.'

During those thirty-five years, forty-five epidemics in all are known to have occurred, and according to facts collected by myself, there have been six more from 1851 to 1873, so that the disease has been epidemic fifty-one times in a space of 58 years. One of those epidemics (1816-18) spread over the whole peninsula and over Sicily, eleven were general throughout whole provinces, twelve had a wide extent within smaller areas, nineteen occurred in single villages, and two in prisons. The seat of the epidemic was oftenest (ten times) the provinces forming the *quondam Kingdom of Naples*, *Lombardy* eight times, the *Campagna* and *Piedmont* each six times, *Sicily* four times, *Venetia* and the *Duchies* each thrice, *Tuscany* twice, and *Liguria* and *Sardinia* each once. I find that the authorities mention as endemic foci of typhus, the Venetian Province of Belluno,[1] the Piedmontese province of Alessandria,[2] the Roman Campagna,[3] Naples, and Sicily.[4]

In the most recent times *France* has enjoyed an almost complete immunity from typhus.

Of sixteen epidemics of typhus which are known to have occurred in that country in the period from 1820 to 1877, six were in the *bagne* of Toulon[5] (1820-29-33-45-55 and -56), one each in the *bagne* of Rochefort[6] (1839), and the prisons of Beaulieu[7] (1827) and Rheims[8] (1837), and there was an outbreak, interesting in more respects than one, and to be spoken of again, in the port of Toulon after the arrival of an Egyptian frigate.[9] Three of the epidemics were connected with the operations of war: in 1823 the pestilence was brought into St. Laurent and Albi by Spanish soldiers from the besieged fortress of Seu-d'Urgel, but it did not spread beyond these towns;[10] in 1856 the troops returning from the Crimean War introduced typhus into Marseilles, Avignon, and other places in the South of France,[11] whence the disease was carried

[1] Facen, 'Gaz. med. Lombard.,' 1850, 217.

[2] Tarchetti, 'Annal. univ. di Med.,' 1871, Oct., 98.

[3] Valentiner, 'Berl. klin. Wochenschr.,' 1870.

[4] De Giacomo, 'De febre per varias Siciliæ plagas popul. grassante,' Catan., 1833, p. 8.

[5] Barallier, 'Du typhus épidém.,' &c., Paris, 1861.

[6] Lesson, 'Revue méd.,' 1839, Juin, p. 458.

[7] Raisin, 'Journ. gén. de méd.,' 1827, vol. 100, 102.

[8] Landouzy, 'Gaz. méd. de Paris,' 1842, p. 376.

[9] Gourrier, 'Relation d'une épid. du typhus observ. à Toulon en 1864,' Montpell., 1866.

[10] Costa, 'Journ. gén. de méd.,' 1824, vol. 87, p. 318; Delbose, ib., 1823, vol. 83, p. 194.

[11] Godelier, 'Gaz. méd. de Paris,' 1856, No. 31 ff; Chauffard, 'Bull. de l'Acad. de méd.,' 1856, Juin.

to Paris and Neufchateau (Vosges),[1] but it did not become widely spread at any of those places; lastly, typhus developed once in the war of 1871, among the population of Metz during the siege.[2] In Paris, at the time of an epidemic of typhoid in 1844, many cases of typhus were observed, but the disease did not assume a properly epidemic character.[3] Finally we meet with two widely-spread epidemics, the one in 1826 in the Arrondissement of Dunkerque,[4] and the other in 1869-71 in the Department of Morbihan;[5] the latter was in connexion with an endemic centre of typhus on the coast of *Brittany*, to which attention has been called only recently, although its presence can be detected, as we now know, in the isolated or slightly epidemic outbreaks that have taken place in the Department of Finistère and the vicinity since 1825.[6] The suggestion that this focus of disease has had its origin in importation of typhus from the opposite English coast, and that it has attained a wider development amidst the truly melancholy hygienic conditions of the district, is a not improbable one. It remains to mention that the disease was imported from the coast to the neighbouring island of Molène, where nearly one-half of the inhabitants sickened of it during the period from September, 1876 to May, 1877.[7]

Switzerland in recent times appears to have been quite free from epidemics of typhus; at least there is an entire absence of trustworthy information about it, excepting a notice of sporadic cases in the autumn of 1844 in the communes of Unter-Affoltern and Regenstorff (Canton Zürich).[8] In *Belgium* during the last sixty years there have been, as far as I can learn, two severe epidemics of typhus, both of them induced by interruption of commerce and failure of crops; the first, and smaller, occurred in 1840-44, particularly in the provinces of Namur, Liege, and Hennegau;[9] the second, and much the greater, was in 1846-48, or at the same time as the general prevalence of the disease in Upper Silesia and elsewhere, and this time it was mostly in the

[1] Garcin, 'Gaz. des hôpit.,' 1856, p. 295.

[2] Michaud, 'Gaz. hebd. de méd.,' 1873, p. 38; Viry, ib., p. 56.

[3] Sandras, 'Revue méd.,' 1845, Fevr. I have not been able to learn anything of a typhus epidemic in Paris in 1852 (Häser, iii, p. 694).

[4] Zandeck, ib., 1826, Dec., p. 504.

[5] Gillet, 'Considér. sur le typhus de Riantes (Morbihan),' Paris, 1872.

[6] Martin, 'Essai sur l'endémicité du typhus dans le depart. du Finistère,' Paris, 1876.

[7] Danguy des Déserts, 'Arch. de méd. nav.,' 1877, Août., p. 100.

[8] 'Bericht des Gesundheitsrathes des Kantons Zürich über das Jahr 1844,' p. 16.

[9] Sauveur, 'Gaz. méd. de Paris,' 1853, p. 203.

provinces of East and West Flanders, scarcely an arrondissement of which escaped the sickness.[1] Whether the disease had been observed at all in Belgium before or after those dates, or to what extent it may have occurred, does not appear from the records before us, inasmuch as the term "fièvre typhoide" is used in the Belgian official reports promiscuously for typhus and typhoid.[2] The same unfortunate ambiguity pervades the professional literature and the official sanitary reports of the *Netherlands*. Rosenstein[3] observes that in Groningen no other form of "typhus" than petechial typhus had occurred during the last fifteen years (1855 to 1868), and he refers to the annual sanitary reports for proof of the frequent occurrence of typhus in the Netherlands; but in these reports, so far as they are before me, typhus and typhoid are thrown together under the single designation "typhous fever,"[4] and the data are of no use therefore for the question at issue. As regards Groningen, the facts observed by Rosenstein, together with the observations from previous years, especially 1816,[5] 1821,[6] and 1854-56,[7] certainly go to prove the endemicity of the disease there; but, besides these, I have found only an occasional trustworthy notice of typhus in the Netherlands, such as that of 1849, when the disease is said to have been prevalent over a great part of the country, and that relating to a small epidemic of typhus in 1867-68 in Overyssel.[8]

In *Denmark* typhus appears to have been of extremely rare occurrence. The sanitary reports, which have been

[1] Mersseman, 'Bull. de l'Acad. de méd. de Belgique,' viii, p. 87; Mareska, ib., p. 123, and ix, p. 513; Warlomont, ib., x, and 'Gaz. méd. de Paris,' 1848, p. 936; ref. in 'Annal. de la Soc. de méd. de Gand.,' xiv, p. 5.

[2] This is true also of the chapter on "Typhus" in the 'Topogr. méd. de la Belgique,' Bruxelles, 1865.

[3] 'Virchow's Archiv,' 1868, Bd. 43, p. 377.

[4] In the summary which Van Capelle (in the 'Nederl. Tijdschr. voor Geneesk.,' 1870, Afd. ii, p. 123) gives of these sanitary reports for the years 1866 to 1868, he expressly states that "typhus en typheuse koortsen" are included promiscuously under the head of "typhus."

[5] Steensma, 'Diss. de typho,' Groning, 1818.

[6] Kymmell, 'Observ. quaed. de typho.' Spec. med., Groning., 1822 (Hospital Report).

[7] 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, p. 196.

[8] Ref., ib., 1868, i, p. 45.

compiled with great care, make mention of only two small epidemics which had prevailed in Fredericia in 1821 and 1839,[1] and of the outbreak in 1843 in the over-crowded prison at Odensee.[2] As to the occurrence of the disease in *Norway* in recent times, there is no reliable information going farther back than 1845.

From 1845 to 1864 the country was quite free of typhus;[3] it showed itself in the latter year, first in the district of Krageröe,[4] and the year after at Christiania, where it broke out first in an inn of the poorest class,[5] and continued in the city and neighbourhood during the years following up to 1872. In the district of Vadsöe (Finmark) typhus attained a considerable diffusion from 1865 to 1871, in company with relapsing fever; it was also prevalent among the fishing population of the Lofoden Islands[6] in 1868, and in 1868-71 in Nordlands-Amt and other districts on the eastern frontier.

These outbreaks of typhus in Norway were undoubtedly connected with importation of the disease from Sweden and Finland, where it had been widely prevalent even at an earlier date; and it is this that explains why its outbreak and epidemic diffusion in Norway took place in the districts nearest to the frontier.[7] In medical writings and official sanitary reports from *Sweden*, we again encounter that hindrance to our inquiry which arises from typhus and typhoid being included in the sanitary returns (up to 1874) under the common designation of "typhous fever."

From the well-known work of Huss,[8] which is subject to the same defect, we learn only this much, that typhus along with typhoid, had been epidemic in Stockholm in 1841-42, 1845-46, and 1851-52. The disease in Sweden became more widely diffused in 1861-70, for which period there are notices of epidemics in Bohuslän in 1861, Hernösand in 1863, and particularly in the northern provinces (Norbotten, Westerbotten, and Wester-Norland),[9] as well as in Götaborg,[10] during

[1] Ref. in 'Bibl. for Laeger,' 1841, iv, p. 333; and Reumert, in 'Sundheds-Colleg. Aarsberetning,' 1855, p. 67.

[2] Hellweg, 'Bibl. for Laeger,' 1845, ii, p. 12.

[3] Boeck, 'Norsk mag. for Laegevidensk,' 1874, iii, R. iv, p. 241.

[4] Homan and Hartwig, ib., 1865, xix, p. 433.

[5] Larsen, ib., 1866, xx, p. 910.

[6] Irgens, ib., 1869, xxiii, p. 513.

[7] Boeck, l. c.

[8] 'Statistique et traitement du typhus et de la fièvre typhoide,' Paris, 1855.

[9] Wistrand, 'Förhandl. ved. Sv. Läk-Sällsk. Sammenkomster. är 1868,' p. 42.

[10] Langell, 'Hygiea,' 1870, xxxii, p. 441.

the famine-years of 1868 to 1870. In the official returns of 1874, typhus is entered by itself, and we find notices of its epidemic prevalence in 1873-75 at Stockholm, 1873-74 in the districts of Uddevalla and Götaborg, and 1875 in Elfsborgs-Län.[1] Isolated cases of sickness from typhus came to notice at various parts of the country in 1876-77.

Whether there are endemic centres of typhus in Sweden can hardly be decided from these scanty data. In the same period when typhus was epidemic in Sweden, there falls a general diffusion of the disease in *Finland*, more particularly in 1865 and in the famine years of 1867-68.[2] *Iceland* also had the typhus widely spread in 1857-61;[3] from Finsen's statements it follows that the disease had unquestionably occurred there often in former times, but had been confounded with typhoid (which is truly endemic in Iceland), while, in these more recent epidemics, both diseases were under observation side by side.

Typhus on *Asiatic* soil has acquired hitherto a very much smaller area of diffusion than in Europe. Apart from its more casual epidemic occurrences in *Asia Minor*[4] and *Syria*,[5] its headquarters are in *Persia*. Polak[6] had already called attention to this fact in describing an epidemic that was prevalent, especially among the troops, in 1857. His statements found support in a paper by Tholozan,[7] who describes the disease as one that is endemic in Persia, and has from time to time overrun a large part of the country in disastrous epidemics, as in the years 1864-65. Tholozan expressly states that typhus rages there under exactly the same circumstances which he had an opportunity of observing in the epidemic among the French troops during the

[1] 'Sundhets-Coll. Berättelse för år 1874,' p. 6; 1875, p. 14. See also Grähs, 'Embetsberättelse för åren 1873-75,' and Warfwing, 'Hygiea,' 1876, xxxviii, p. 373.

[2] Palmberg, ib., 1870, xxxii, p. 68; Estlander, 'Archiv für klin. Chirurgie,' 1870, xii, p. 453; Hjelt, 'Nord. med. Arkiv,' 1870, ii, p. 26.

[3] Hjaltelin, in 'Sundhedscoll. Aarsberetn. for 1855,' p. 67, and 'Edin. Med. Journ.,' 1862, Sept., p. 214; Finsen, 'Jagttagelser angaaende Sygdomsforhold i Island.,' Kjöbenh, 1874, 15.

[4] West, 'New York Med. Record,' 1869, March, p. 27.

[5] Post, ib., 1868, June, p. 149; Robertson, 'Edin. Med. and Surg. Journ.,' 1843, April, p. 247.

[6] 'Zeitschrift der Wiener Aerzte,' 1859, p. 138.

[7] 'Bull. de l'Acad. de méd.,' 1866, xxxi, p. 333.

Crimean War. *India*,[1] *Further India*,[2] and the *East Indian Archipelago*,[3] appear to be quite free from typhus, and, according to Wernich,[4] the disease is quite unknown in *Japan*. On the other hand, there are trustworthy accounts[5] of its endemic prevalence in the north of *China*, especially in Pekin, where the disease is met with every year in the winter season among the poorer of the populace, and where it broke out in disastrous epidemics in the years 1864-66. On the continent of *Australia*, in *New Zealand*, and in *Polynesia*, there has been complete immunity from typhus hitherto; into *Tasmania*[6] it has been introduced several times by ships, but it has been prevented from spreading at all widely over the island by strict precautionary measures.

Nor does typhus appear to occur throughout the greater part of the continent of *Africa* and the islands belonging to it, this exemption being alleged particularly for *Cape Colony*,[7] for the *East Coast* and *Madagascar*,[8] and for the *West Coast*. On the other hand, the description given by Griesinger[9] of "simple typhus," observed by him among the natives of *Egypt*, points to it having been the disease that we are now considering, and the absence of an exanthem, which Griesinger emphasises, must be explained by the difficulty of

[1] The assertion of Morehead ('Clinical Researches,' i, p. 307), that typhus is unknown in India has been confirmed in the latest publications by Chevers ('Med. Times and Gaz.,' 1879, Aug., p. 121), and other Anglo-Indian physicians. The epidemic of 1859-61 in the N. W. Provinces and the Punjaub, described by Walker ('Edin. Med. Journ.,' 1861, May, p. 986), is erroneously designated as one of typhus; it is there an affair of relapsing fever and bilious typhoid, which we shall enter upon in the next chapter.

[2] Breton ('Considér. sur la guérison des plaies . . . chez les Annamites,' Par., 1876, p. 10) and Danguy des Déserts (Considér. sur l'hygiène de l'Européen en Cochinchine,' Par., 1876, p. 12) mention, under the name of "fièvre des bois," a disease occurring in Anam, which corresponds to typhus, according to their reading of it; but I do not regard this conclusion as trustworthy.

[3] Heymann ('Schmidt's Jahrb. der Med.,' 1846, Juli, lii, p. 96), had not seen a single case of typhus during twenty-five years' practice in Java and Sumatra.

[4] 'Deutsche med. Wochenschrift,' 1878, p. 101.

[5] Morache, 'Rec. de mém. de méd. milit,' 1866, i, 142, and 'Annal. d'hyg. publ.,' 1870, Jan., p. 57.

[6] Hall, 'Transact. of the Epidemiol. Soc.,' 1866, ii, p. 69.

[7] Egan ('Med. Times and Gaz.,' 1872, Jan., p. 111) says that he had seen two cases of typhus (?) at Williamstown in the period from 1857 to 1872.

[8] Davidson, ib., 1868, Dec., p. 646; Borchgrevink, 'Norsk Mag. for Laegevidensk,' 1872, Tred. R., ii, p. 247.

[9] 'Archiv für physiol. Heilkd.,' 1853, xi, p. 358.

detecting it on the skins of the dark-coloured races. This conjecture appears to be made all the more probable by the fact communicated[1] to Griesinger by Penay, that typhus is endemic in *Nubia*, and often very malignant, and that the exanthem comes out very clearly there in persons with whiter skins. Further, Veit[2] speaks of a severe epidemic of typhus at Cairo in 1836. From *Tunis* there is a record of a great typhus epidemic in the famine-year of 1868[3]; from *Algiers*, where typhus is probably endemic in Kabylia, becoming epidemic from time to time under peculiarly unfavorable states of hygiene,[4] there are accounts of a general epidemic diffusion in 1861-62;[5] further of an epidemic in 1863 among an Arab tribe in the district of Setif (Prov. Constantine),[6] and finally of the disastrous outbreak that took place in that country, as well as elsewhere, during the famine-year of 1868.[7]

For the *Western Hemisphere*, the earliest information as to the occurrence of typhus comes from *Mexico* and *Peru*. According to the perfectly reliable statements of Francesco Bravo,[8] the disease, which he speaks of under the name *tabardete* then current in Spain, broke out in Mexico for the first time in 1570 on importation from Spain; and it can hardly be doubted that the pestilence known under the vernacular name "matlalzahuatl," which was prevalent in 1576, 1736, and 1762 in the interior of Mexico[9], on the table-land as well as in the mountains, was typhus; for Montana[10] tells us that in the epidemic of typhus in the City of Mexico, in 1813, the natives used that name to designate the disease, recognising in it the malady which they had known well in former times. Our later information about typhus in Mexico

[1] Ib., 374. I shall afterwards have occasion to mention a few interesting facts about the repeated importation of the disease into European ports by Egyptian ships of war (p. 586).

[2] Württemb. med. Correspondenzbl.,' 1851, p. 313.

[3] Ferrini, 'Annali univ. di med.,' 1869, Maggio, p. 241.

[4] Léonard et Marit, 'Rec. de mém. de méd. milit.,' 1863, Août, p. 81.

[5] Vital, ib., 1869, Févr., p. 81. [6] Léonard, l. c.

[7] Vital, l. c.; Perrier, ib., 1869, Juin, p. 449, 1870, p. 461; Challan, 'Gaz. méd. de Strasb.,' 1868, No. 21; Arnould, 'Gaz. méd. de Paris,' 1869, No. 46 ff., 1870, No. 2 ff.; Batterel, 'Étude sur quelques cas de typhus, &c.,' Paris, 1872.

[8] 'Opera medicinalia,' Mexico, 1570. Compare Villalba, 'Epidemiol. Españ.,' i, p. 186.

[9] Humboldt, 'Voyage en Amérique,' Part iii, p. 750.

[10] 'New York Med. Reposit.,' 1819, v, p. 1.

comes from French physicians,[1] who had opportunities of observing the disease there during the war and subsequently; they all agree in stating that it is confined to the *tierra fria*, and that there it is endemic. To *Peru* also, typhus was probably imported from Spain at a very early period, for it is universally known there under the Spanish name *tabardillo*.[2] The disease has been observed a few times as war-typhus, for example at Lima in 1821 during the investment of the city,[3] and at Callao in 1825-26 under the same circumstances; but it has been especially in the Sierra region that it has raged, making great ravages among the Indians, most recently in 1853-57.[4]

Of *Chili*, Pöppig[5] has already remarked that, if acute diseases are in general less frequent in that country than in other parts of the globe, that does not apply to "typhus;" and the most recent accounts by Lantoin[6] and Boyd[7] make it clear that he means the disease which we are now considering. Lantoin, whose observations relate to the Quebradas (narrow river valleys) in the north of the country, expressly states that the disease corresponds to the typhus of the English physicians, differing from typhoid in the exanthem and in the absence of the intestinal lesion; Boyd, in like manner, says that "it corresponds exactly to the typhus fever of Ireland;" while both describe it as a malady of frequent occurrence in Chili, and not unfrequently epidemic. The writings of Sigaud[8] and Dundas[9] make it probable that *Brazil* is not exempt from typhus, but they

[1] Poncet, 'Rec. de mém. de méd. milit.,' 1863, Févr.; Briault, ib., 1864, Mars.; Coindet, ib., Mai; Wuillot, 'Presse méd. Belge,' 1866, No. 40, p. 314.

[2] Tschudi, 'Oest. med. Wochenschrift,' 1846, No. 12.

[3] Smith, 'Edin. Med. and Surg. Journ.,' 1840, April, p. 332, 1842, April, p. 362. This author is not clear as to the nature of the epidemic, nor of the pestilence among the Indians (Epidemiol. Trans.,' 1863, i, p. 222); the latter he considers to have been yellow fever.

[4] Macedo, 'Gaz. med. de Lima,' 1858, No. 48; Archd. Smith, 'Transact. of the Epidemiol. Soc.,' 1863, i, p. 222.

[5] 'Clarus und Radius, Beiträge zur pract. Heilkd.,' 1834, i, p. 528.

[6] 'Arch. de méd. nav.,' 1872, Mars., p. 165.

[7] 'Edin. Med. Journ.,' 1876, Aug., p. 115.

[8] 'Du climat et des malad. du Brésil,' Paris, 1844, p. 251.

[9] 'Sketches of Brazil,' Lond., 1852, p. 222. Murchison, who denies the occurrence of typhus in South America at all, takes the facts given by Dundas as relating, not to typhus, but to "adynamic remittent fever." I am not clear as to his grounds for this opinion.

give us no means of estimating the extent to which it prevails. From *Central America* I know of only one reliable piece of information about typhus, which relates to an epidemic in Nicaragua in 1851.[1] In the *West Indies*, typhus does not appear to have been seen at all in recent times. Wright,[2] whose information dates from the end of last century, says that the disease was not rarely introduced at that period by drafts of troops from England; but, in the English 'Army Reports' of the present century, from these colonies and the Bermudas, I find that only two cases of typhus occurred in the Bermudas from 1817 to 1836,[3] and only four from 1837 to 1846,[4] while, in the West Indies from 1816 to 1836 there were only forty-eight cases.[5] In later reports, down to 1879, the disease is not mentioned at all; and in the numerous notices before me from the West Indies, of a medico-topographical and epidemiological kind, there is not a word said of typhus.

Typhus came to the *United States* and to *British America* much later than to Mexico, and in those countries it has never attained the same importance as in the latter or on the continent of Europe. The epidemiological references by American physicians in the eighteenth century give no indication that they had ever seen typhus, and as late as the first twenty years of the present century the disease had been at any rate rare with them. The proper era of typhus for the United States and Canada begins with the period when emigration from Ireland had set in on a large scale.[6]

[1] Bernhardt, who furnishes the information on this epidemic ('Deutsche Klin.,' 1854, No. 8), remarks that the disease was of quite the same type as that which he saw in Upper Silesia in 1847.

[2] 'Duncan's Annals of Med.,' 1797, ii, p. 345.

[3] 'Statist. Reports,' 1839, p. 6*b*.

[4] Ib., 1853, p. 176.

[5] Ib., 1838, p. 7.

[6] Wynne, 'Amer. Journ. of Med. Sc.,' 1852, April, 417; Drake, l.c., p. 430. To show the enormous extent to which typhus has been imported into North America in recent times, I give the following figures: whereas, from 1833 to 1847, 291 typhus patients were received into the hospital of Quebec, out of an immigration averaging 23,000 per annum, the number rose in 1847 to 8574 among 98,000 immigrants, besides 5000 persons who died on the way, mostly from typhus. In the hospital on Staten Island (New York), nearly 3000 immigrants sick with typhus were received the same year; and on Deer Island (Boston Harbour) several thousands of typhus cases were observed among the arrivals during the same period.

We thus explain the fact that the ports on the east coast of North America have been the headquarters of the disease, and that the largest contingent of the sick has been supplied by the immigrants themselves, or their countrymen with whom they had come in contact. In many cases, the disease was confined exclusively to immigrants; in others it spread among the population of the seaports, particularly in the filthy and crowded quarters, but without ever attaining the dimensions of a great epidemic. Thus we find that typhus has been repeatedly imported since 1820 into Canada, where the cities of Quebec and Montreal lying directly in the track of the immigration have suffered most. From time to time, it is true, the disease has travelled along the banks of the St. Lawrence into the interior, but it has not become widely diffused therein.[1] From Halifax (Nova Scotia) also, there is an account of importation of typhus by Irish immigrants in 1827, and of the sickness spreading among the inhabitants of the town itself.[2] There is information from Boston of the introduction of the disease by emigrants in 1838 and 1847,[3] and from New York for 1818, 1825, 1837, 1848, and 1852; among the population of the latter city typhus spread considerably in 1818, 1827, 1837, and 1847, so that, while the deaths from typhus (and typhoid) in 1846 were only 256, they rose in 1847 to more than 1400.[4] The introduction of typhus by immigrants into Philadelphia is attested by writings of the years 1827 and 1847; in the proletariat quarter of the city, it was widely prevalent as an epidemic in 1836.[5] Several importations have taken place into Hartford, Conn., without any extension to the populace;[6] in like manner, at New Orleans in 1847, it remained limited to the immigrants, and to the doctors, nurses, and others in

[1] Fraser, 'Brit. Amer. Journ. of Med. and Phys. Sc.,' 1848, iii, p. 61; Douglas, ib., p. 261; Badgley, ib., 1849, iv, p. 88.

[2] Donelly, 'Lond. Med. and Phys. Journ.,' 1829, July 11.

[3] Gerhard, 'Amer. Journ. of Med. Sc.,' 1837, Feb., p. 289; Clark, 'Ship Fever,' &c., Boston, 1850; Upham, 'New York Journ. of Med.,' 1852, March.

[4] Brown, 'Amer. Med. Recorder,' 1828, July 1; Reese, 'New York Journ. of Med.,' 1847, May; Smith, 'Transact. of the Amer. Med. Assoc.,' 1848, i, p. 109; Schilling, 'New-Yorker med. Monatschrift,' 1852, i, No. 8.

[5] Gerhard, l. c.; Sargent, 'Amer. Journ. of Med. Sc.,' 1847, Oct., p. 529.

[6] Russell, 'Proc. of the 63rd Annual Conv. of the Connecticut Med. Soc. 1855.

the hospitals; and the same holds good for the typhus imported into Baltimore in 1850.[1] The disease was remarkably frequent in 1863-64 in New York, Philadelphia, and other places in the Eastern States.[2] On the other hand, it is a noteworthy fact that the most careful search among the plentiful epidemiological records in the literature of the United States, fails to discover a single statement as to the occurrence of typhus in the Mississippi Valley or in the Western States; so that the greater part of the continent appears to enjoy absolute immunity from the disease, and in no part of the whole territory do endemic centres of typhus appear to have formed, notwithstanding importations on a large scale.

§ 134. Mostly a Disease of the Temperate and Cold Zones.

The history of typhus, as we have said, is the history of human misery. With the exception of the plague and of relapsing fever, which latter connects with typhus very closely in respect of its epidemiology and its etiology in general, none of the acute infective diseases has been so closely associated with *definite* nuisances that have arisen out of a miserable hygiene; none stands in its genesis so *directly* under the influence of this etiological factor; and even the influence which conditions of climate, season, and weather exert upon the occurrence and amount of typhus, may be ascribed not less (but perhaps even more) to the habits of living of the populace as adapted to the climate and season, and to the varying produce of the soil as dependent on the weather, than to any direct action of the latter upon the specific cause of the disease.

It follows from the prevalence of the disease in Persia, and the epidemics that have been observed in Tunis, Algiers, and Nicaragua—to mention only well-established facts—that the doctrine which used to be held, of the palm-zone

[1] Wynne, l. c.

[2] Da Costa, 'Amer. Journ. of Med. Sc.,' 1866, Jan., p. 17; Corse (ib., p. 158) says that, while the mortality from typhus at Philadelphia was only 37 in 1862, it rose in the two following years to 131 and 335 respectively.

and regions with an isotherm of 68° F. (20° C.) and upwards enjoying a complete immunity from typhus, is not universally applicable. But it is nevertheless a remarkable thing that great regions of the tropics, such as the East and West Coasts of Africa, India, the East Indies, and the South of China, have remained hitherto quite exempt (or at least seem to have so remained), and that, in other tropical countries, such as Mexico and Peru, it is particularly the lofty plateau (the *tierra fria*) and the Sierra region that have been the seat of the disease. Still, we are taught to be cautious in generalising, by the recent experiences of the occurrence of typhoid (typhus abdominalis) and relapsing fever in India and other tropical regions which we had considered to be exempt from those diseases also. In fact, as we have said already, it is an open question how far the protection which the tropics seem to enjoy from typhus fever in virtue of their peculiar climatic conditions is a direct one or an indirect—that is to say, one that is involved in the mode of living among the people as adapted to the climate. At all events, the influence of climate in general cannot well be ignored, inasmuch as the amount of sickness stands in an unmistakeable ratio to particular seasons of the year in those parts of the globe which are the permanent seats of typhus.

§ 135. Connexion with Cold Weather.

Out of 147 epidemics of typhus which have occurred in the temperate or cold latitudes of the Eastern and Western Hemispheres, and of which we have accurate data as to time of their outbreak and their course, 30 reached their acme in spring, 28 in winter and spring, 21 in spring and summer, 19 in summer and autumn, 18 in autumn, 17 in summer, and 14 in autumn and winter. This result corresponds to the opinion of most of the authorities on typhus in Britain[1] as to the prevalent season of the disease, the maximum

[1] See the 'Report on the Sickness among the Troops in the U. K.,' 1853, p. 17, 33; Alison, in 'Reports on the Sanitary Condition of the Labouring Population of Scotland,' Lond., 1842; Thomson, 'Edin. Med. and Surg. Journ.,' 1838, July, p. 87; Craigie, ib., 1834, April, p. 257; Henderson, l. c.; Percival, l. c.; Hamilton, 'Lancet,' 1867, Nov., p. 608.

being in winter and spring, as well as to the conclusions of Dehse for Dorpat, Marroin for Constantinople, Tommasi[1] for Naples, and Morache for Pekin. Also the epidemics that are known to have occurred in Tunis, Algiers, Persia, and Peru, have all been prevalent in spring, or in winter and spring. This fact is further borne out by statistics of the deaths from typhus in England, and of the admissions for typhus into several British fever hospitals.

Table of Deaths from Typhus in England and Wales.

Year.	Winter.	Spring.	Summer.	Autumn.
1838	5813	4980	3826	4155
1839	4236	3733	3278	4419
1840	4798	4164	3726	4460
1841	3941	4218	3498	3197
1842	5131	3910	3480	3680
	23,919	21,005	17,808	19,911

Table of Admissions for Typhus into Hospitals, for each month of the year.

Hospital.	Period.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.
London Fever Hospital[2]	1848–62	533	460	616	631	636	499	435	393	322	335	342	313
Dundee Infirmary[3]	1859–66	251	156	209	204	205	167	178	166	195	234	282	245
Glasgow Infirmary[4]	1871–79	33[5]	29	33	27	34	28	20	18	27	31	35	39

[1] 'Il Morgagni,' 1868, p. 23.

[2] Tweedie, 'Lancet,' 1860, Jan.; Murchison, p. 52.

[3] Maclagan, 'Edin. Med. Journ.,' 1867, Aug., p. 143.

[4] Macphail, 'Glasg. Med. Journ.,' 1879, Oct., p. 257.

[5] The figures give the average number of admissions for each month during those years.

If, then, there can be no doubt that the maximum of typhus cases falls distinctly in the colder months (winter and spring), yet it is proved by the experience of the severe pestilences of 1816-19 in Italy, of 1817-19 and 1847-48 in Ireland, and of 1847 and 1856 in Upper Silesia, as well as by the course of the minor but protracted epidemics of 1819-24 in Westphalia, 1838-39 in Dittmarschen, and 1837-38 in Glasgow, that typhus is quite independent of *season* and *weather* in its development and epidemic diffusion, that neither the upper nor lower extremes of temperature or moisture can further or hinder the disease in its breaking out and progress, nor any other condition of weather exert a perceptible influence in these respects. It is in this sense that almost all the authorities express themselves for just those regions which are most frequently visited by typhus—for Britain,[1] Italy,[2] Upper Silesia,[3] and other countries.

§ 136. Independent of Telluric Conditions.

There is not the smallest reason, deducible from the facts, for supposing that *characters of the soil*, such as *elevation, conformation, kind of rock, saturation*, and *amount of organic detritus*, exert any influence whatsoever upon the origin and diffusion of the disease. The disease has been equally prevalent and equally severe on plateaus and among mountains as in basins, on dry as on wet soil, on light sand as on heavy clay, on porous rock as upon firm; it has nowhere and at no time shown any particular affinity for that quality of surface which is usually designated *malarious soil*. Ireland, which is unquestionably the worst typhus centre in Europe, enjoys almost absolute immunity from malarial diseases. Again, while the maximum of typhus cases falls in those seasons in which influences of the soil in general, and malarial influences in particular are least felt, the minimum is found to be in the autumn months, which are the proper malarial season.

[1] Harty, Duncan, O'Brien, Cheyne, Graves, ll. cc.; Corrigan, 'Lancet,' 1830, June; Hunter, 'Edin. Med. and Surg. Journ.,' 1819, April, p. 234; Mateer, 'Dubl. Journ. of Med. Sc.,' 1836, Sept., p. 34; Murchison; and various others.

[2] Ramazzini, 'De constitutionibus dissert,' i, in Opp. Londini, 1718, p. 106; Omodei, i, p. 13; Tarchetti.

[3] Frank, 'Die Berichterstatter aus der Epidemie, 1876-77.'

"For a considerable period," says Graves,[1] "there was a great tendency among physicians to refer the origin of typhus, and almost every variety of fever, to malaria, or unwholesome emanations from the soil, produced by the decomposition of vegetable matter. In Ireland, facts do not bear out this hypothesis, for, as already stated, when an epidemic of fever has become established, it breaks out simultaneously in situations the most different, and in some where no such emanations can be supposed to exist. Thus I have seen a whole family affected in "the telegraph," situated at the summit of Killiney, a mountain formed of bare granite; and, indeed, the granite and mountain districts beyond Rathfarnham, Tallaght, and Killikee supply the Meath Hospital with its worst cases of typhus." Rogan, Corrigan, and other Irish physicians had previously expressed the same opinion. Observations instituted in Italy on the points in question have led to a similar conclusion: "Gli è poi rimarchevole," says Mantovani,[2] "come in tale invasione il morbo prediletti non abbia i terreni paludosi e malsani, e sembri anzi minacciare quasi davvantaggio i più alpestri e celebrati per la purità dell' aria e del clima. Un distretto di 20 e più comuni (Rosato) immersi quasi tutti fra le risaie andava incolume, quando il restante della provincia pavese ne era bersagliato più e meno." The arguments adduced by Rosenstein[3] in favour of a connexion between typhus and malarial disease do not appear to me to be relevant in any respect; they are that typhus predominates in low grounds (as on the malarious soil of Upper Silesia), that the two diseases coincide in time, that typhus not unfrequently begins with true intermittent paroxsyms, and passes into intermittent fever towards the end of the process, and finally that typhus occurs side by side with relapsing fever.

§ 137. Epidemic Typhus Coincident with a Time of Want.

When we inquire into the influence exerted by the state of social well-being on the production of typhus, there is always one fact forcing itself into the foreground, which has arrested the chief attention of observers at every period, viz. *the coincidence in time between epidemics of typhus and the state of want brought about by failure of the crops, commercial crisis, war, and other far-reaching calamities.* Almost all the severe typhus pestilences of past centuries afford striking evidence of this, as the foregoing historical sketch will have shown; also the epidemics of recent times—those of 1847-48 throughout a large part of Europe, the epidemic spread of the dis-

[1] 'Clinical Medicine,' Dublin, 1843, p. 42.
[2] 'Annali univ. di med.,' 1817, Agorto, p. 138.
[3] 'Virchow's Archiv,' 1868, Bd. 43, p. 379.

ease in Ireland in 1817-19, 1821-22, 1836-38, and 1862-64, that of 1816-18 in Italy, of 1856-67 and 1876-77 in Upper Silesia, of 1868-69 in East Prussia, Sweden, and Finland, and simultaneously in Tunis and Algiers—all these outbreaks, not to mention the more circumscribed epidemics, which have also been in many cases associated with want, furnish indisputable proofs that there subsists some causal connexion between the disease and the state of want. This connexion has been variously interpreted from various sides. Some observers rate it so highly as to see in the typhus the direct consequence of want. Bateman[1] was the first to give definite expression to the opinion "that deficiency of nutriment is the principal source of epidemic fever;" Corrigan entitled his well-known essay "Famine and Fever as Cause and Effect,"[2] and so the notion of "hunger typhus" (*typhus famelicus*) became finally adopted. The first point that has to be urged against this conception is that "hunger sickness" by no means corresponds to a single pathological idea; on the contrary, it is compounded of a somewhat extensive series of local affections of various kinds, including particularly gastro-intestinal catarrh, dysentery, disorders of nutrition, extreme degrees of emaciation, scurvy, dropsy, and the like; typhus is associated with them, and not unfrequently typhoid also; but typhus first develops when other etiological factors have been added, as several of the more recent observations go to show. Further, it tells against the assumption of a specific importance for the production of typhus residing in this disease-factor, that states of great want have been endured for a long time and have given rise to manifold diseases—to hunger-sickness in the true sense—without typhus making its appearance.

The observations published by Virchow[3] on the ill health of the poverty-stricken population of Spessart at the time of the prevalent scarcity there in 1852, are of especial interest in this respect. With reference to the epidemic of typhus in 1856 in the circle of Pless (Upper Silesia) Frank[4] gives a decided negative to the question "Was the typhus engendered by the famine preceding it?" and he points out that "all

[1] L. c., p. 4.

[2] 'On Famine and Fever as Cause and Effect in Ireland,' Dubl., 1846.

[3] 'Verhandl. der Würzb. phys.-med. Gesellschaft,' 1852, iii, p. 157.

[4] L. c., p. 424.

the villages to the west and north of that circle remained free from the epidemic, although they were equally under the scourge of want and misery." In Ireland and in Italy there have been famine-years over and over again, without epidemics of typhus; and, what is especially noteworthy, India has been visited of recent years by the most frightful famines, counting their victims by millions, but typhus has never grown into an epidemic there.

Again, there is no inconsiderable number of observations showing that the disease may develop quite independently of want, or, at any rate, that the cases of typhus can by no means be brought into direct relation with that state.

"In my report," says the experienced Graves,[1] "of the fever which devastated the West of Ireland in 1822, I advanced the opinion that such epidemics are brought on by a great dearth of provisions, and their unwholesome quality. These are no doubt aggravating circumstances, but that they are not the sole or even the chief causes of typhus epidemics is evident from what I have since frequently witnessed, viz. the occurrence of fever epidemics during years of plenty, of which 1826 was a remarkable example." Weiss (l. c.) shows that there could be no question of famine, not even remotely, in the endemic occurrence of the disease in the circle of Flatow, or in the epidemic of 1860 in particular. In like manner, Graetzer[2] says of the Breslau epidemic of 1868-69, that want and privations could in no wise be accused of being factors in the etiology. Maclagan[3] points out that trade and manufactures were flourishing in Dundee at the time of the typhus epidemic of 1865-66, and that the workpeople who flocked to the town in large numbers, and among whom typhus afterwards developed to a considerable extent, obtained good wages, and were certainly not suffering from want. This proves, says Maclagan, that it was not want and misery, but another factor, namely, the overcrowding in the workmen's dwellings, that gave rise to the disease. I shall give later on the more important of the observations on this head that have been made in Algiers and Tunis.

Famine and typhus, then, have no necessary connexion as cause and effect, as Virchow[4] has already pointed out in his history of the typhus epidemic of 1847-48 in Upper Silesia. The typhus poison, it is clear, finds a particularly suitable soil wherein to develop and acquire potency in a populace reduced by hunger; but that detriment only amounts to a material predisposing factor of disease,[5] and it will make itself

[1] 'Clinical Medicine,' p. 41. [2] L. c., p. 34. [3] L. c., p. 140.

[4] 'Virchow's Archiv,' ii, p. 276.

[5] "If want by itself produces no typhus," says Virchow ('Ueber den Hungertyphus,' Berl., 1868, p. 37), "still it makes men in a high degree ready to take in the germs of the disease and to let them develop."

felt all the more where other lowering causes have been reducing the power of resistance in the individual at the same time.

§ 138. Associated with Filth, Overcrowding, and Privations.

A glance at the countries or regions of Europe which are still noted, even in the most recent times, as permanent seats of typhus, shows that, whatever be their differences in climate, and in terrestrial and national characters, there is a remarkable agreement among them in respect of certain *blots in their social well-being*. In these factors therefore we are led to find the essential cause of the disease being prevalent as an endemic ; and that assumption receives actual confirmation in the circumstance that the epidemic outbreaks of typhus in these countries always go hand in hand with an aggravation of such hardships, and that, at other points of the globe where there is no endemic typhus, the disease is most apt to become epidemic whenever these injurious influences make themselves felt to any considerable extent. It is always and everywhere the wretched conditions of living, which spring from poverty and are fostered by ignorance, laziness, and helplessness, in which typhus takes root and finds nourishment; and it is above all in *the want of cleanliness, and in the overcrowding of dwellings, that are ventilated badly or not at all and are tainted with corrupt effluvia of every kind*. The prototype of these conditions is found in Ireland, which is the greatest sufferer from the disease; all observers agree that in them lies the true cause of typhus, unconquerable in and inseparable from the Irish proletariat, faithfully following the Irishman wherever he transplants himself and his misery.

"Crowded, filthy, ill-ventilated apartments," says Harty,[1] "and neglect of personal cleanliness [are the causes which] more immediately lead to the formation of the fever;" and he goes on to say: "the causes which render this country prone to discontent and liable to the frequent visitations of fever, are the wretched condition and consequent habits of its lower orders, both artisans and labourers, and this immediate cause

[1] L. c., p. 146.

is owing to their want of employment, of education, and of moral culture," the more remote causes of this melancholy state of things being given as "deficiency of capital, absence of the chief landed proprietors, exorbitant rents, and minute subdivisions of the land." Popham's opinion is to the same effect:[1] "It must be admitted that most of the stimuli which quicken the seeds of fever exist among the lower classes of Irish—deficient and bad food, intemperate habits, neglect of personal cleanliness, and *the effluvia accumulated from numbers herding together like gregarious animals, in ill-ventilated dwellings*. These hurtful habits the Irishman does not leave behind him when he emigrates." This is the view that is held also by Rogan,[2] Speer,[3] Babington,[4] Davidson,[5] Maclagan,[6] and others. Schilling, in his report (l. c.) on the outbreak of typhus in 1852 among the emigrants collected on Ward's Island, New York, points out that almost all the cases came from the crowded depôts which the commissioners of emigration in New York had erected for the reception of these unfortunates, and that 75 per cent. of them were Irish. Arnott,[7] Alison,[8] Paterson,[9] and others have described how typhus broke out among the Irish who had wandered to Scotland and lived there amidst their filth and misery, and how it spread from these circles to the Scottish working classes.

We meet with precisely the same un-hygienic wretchedness in the districts of Russia[10] which are ravaged by endemic typhus, in Upper Silesia, in certain districts of East and West Prussia, in Italy,[11] as well as in the more recently discovered focus of typhus in Brittany; in all those localities the disease is proportionate, both in its extent and in its severity of type, to the height which social wretchedness has reached, varying with the increase or abatement of the latter.

The faithful description which Virchow has given of the conditions of living among the population of Upper Silesia, mostly Poles, concludes with these words: "Everyone must have carried away the conviction that the state of the people in Upper Silesia is too horribly

[1] 'Edin. Med. and Surg. Journ.,' 1853, July, p. 53.

[2] L. c., pp. 18, 78, 81.

[3] 'Dubl. Hosp. Rep.,' 1822, iii, p. 198.

[4] 'Dubl. Journ. of Med. Sc.,' 1837, Jan., p. 404.

[5] L. c., p. 57.

[6] L. c., p. 140.

[7] 'Report on the Sanitary Condition of the Labouring Population of Scotland,' Lond., 1841.

[8] 'On the Epidemic of 1843 in Scotland,' &c., Edin., 1844.

[9] 'Edin. Med. and Surg. Journ.,' 1848, Oct., p. 379.

[10] See the accounts by Erdmann ('Med. Topogr. des Gouvernement und der Stadt Kasan,' Riga, 1822, p. 251), and by Auer, Oesterlen, Behse, and others.

[11] See Palloni, l. c., i, p. 25; and Corradi, 'Annali,' iv, p. 715, *seq.*

wretched for a writer to say even a few words about it, without suggesting to a stranger that the description must be exaggerated."[1] The observations here referred to were made in 1848, but they are confirmed by the reports of subsequent epidemics in the same province; and the same tone pervades the accounts of the social condition of those circles in East[2] and West[3] Prussia, Westphalia,[4] and other provinces which have had repeated visitations of typhus, as well as the accounts given by Gillet and Martin of the typhus-centres in Brittany. "In the Breton departments," says the latter (p. 43), "sanitation has been hitherto a dead letter, and it will remain so for a long time to come."

A very interesting supplement to this theme is furnished, as we have already indicated, by the history of the typhus epidemic of 1868 in Algiers. In consequence of extreme want, a frightful plague of famine broke out among the Kabyles; the unfortunates flocked in crowds to the towns in search of food, and the authorities did all they could to help them; buildings were run up to receive them, which were quickly overcrowded, and it was not until then that typhus broke out, spreading quickly to the residents of the towns. "It is to be remarked here," says Batterel[5]—and the other authorities, Challan, Périer, and Arnould agree with him—"that none of the starving people had typhus up to then; it was undoubtedly their crowding together that favoured the hatching of the typhus germ which they carried in their rags." Under the same circumstances, typhus sprang up in Tunis in 1868, according to the statements of Ferrini, and at St. Gall in 1817, according to Rheiner.[6] In the latter case, there was extreme want from stagnation of trade following a commercial crisis; the starving country people flocked into the town in a body—"agmina hominum, facie lurida, cavis oculis, penitus emaciato corpore," as the narrative has it (p. 12)—so that there was very soon a state of excessive overcrowding in the houses and hospitals where the unfortunates had been taken in, and a severe epidemic of typhus quickly sprang up.

Another group of typhus pestilences, interesting in the same connexion, are the outbreaks among *colonies of workmen*, who had been brought together in hundreds or in thousands at particular spots, in order to construct earthworks, or roads, or railways. Coming under the harmful influences of a crowded life in filthy rooms without any

[1] 'Virchow's Archiv,' ii, pp. 162—167.

[2] Passauer, l. c., p. 83; Müller, l. c., p. 6.

[3] Weiss, l. e.; Neumann, l. c.

[4] Nicolai, l. c.

[5] L. c., p. 6.

[6] 'Diss. sistens observ. in topogr. med. urbis Helvet. Sangalli, et t phos. epid. ibi annis 1817 et 1818 existentem,' Tubing., 1818.

ventilation, in barracks temporarily run up for them, or in the miserable huts erected by themselves, or even in damp holes in the earth, they have furnished the starting points of severe typhus epidemics.

One of the best known instances, because the most recent, is that of the workmen who flocked together in 1867-68 to make the railway in East Prussia,[1] and another instance occurred almost at the same time among the road-makers in the circle of Franzburg, department of Stralsund.[2] Outbreaks of typhus under the same circumstances as in East Prussia had occurred before, particularly in 1844 in the circle of Gratz[3] and 1846 in the circle of Cilli,[4] and in 1845-46 in the Government of Novgorod.[5]

The etiological significance of the factor above referred to comes out very strikingly in *the outbreaks of typhus in overcrowded prisons, work-houses,* and similar institutions.

There are the well-known facts relating to the "black assizes" at Cambridge[6] in 1522, at Oxford[7] in 1577, at Exeter[8] in 1586, at Taunton[9] in 1730, at Launceston[10] in 1742, and at the Old Bailey in London[11] in 1750, in which the typhus that had taken origin in the crowded jails was communicated by prisoners, during the sitting of the Court, to judges, jurymen, and the public.[12] Harty[13] points out, with reference to the outbreaks of typhus in the Dublin jails, that the disease was very rare in those prisons that were used for the reception of criminals immediately before the assizes, in which they were detained only a short time, and in which there was accordingly never any overcrowding, or not for long; while, in the prisons set apart for criminals sentenced to transportation, which were not unfrequently so crowded that eight or ten persons had to live for a considerable time in a cell meant for three, typhus broke out whenever that degree of crowding was reached. Other examples of the development of this kind of prison fever, the "jail fever" of the

[1] See particularly Passauer, p. 76.
[2] V. Treskow, l. c.
[3] Kicker, 'Oest. med. Wochenschrift,' 1847, p. 161.
[4] Ferstler, 'Oest. med. Jahrb.,' 1847, März, p. 287.
[5] Bardowski, 'Med. Ztg. Russl.,' 1850, p. 172.
[6] Ward, 'Philos. Transact.,' 1758, vol. 50, p. 703.
[7] Id., p. 699.
[8] Hollingshed, 'Annals,' ii, p. 1547.
[9] 'Gentleman's Magazine,' 1750, May.
[10] Huxham, 'Observ. de aëre et morbis contagiosis,' Lond., 1752, ii, p. 82.
[11] Pringle, 'Observ. on the Diseases of the Army,' Germ. transl., Altenb., 1772, p. 392.
[12] [In some of these cases, if not in most of them, the prisoners were not suffering themselves from typhus.]
[13] L. c., p. 159, Append., p. 264.

English, are as follows: in the prisons of Prague[1] in 1823, in the police prison of Vienna[2] and the military prison of Tarnopol[3] in 1867, in the prison of Posen[4] in 1828, in several prisons of the Palatinate[5] (Kaiserlautern, Zweibrücken, &c.) in 1853-55, in the penitentiary at Odensee[6] in 1843, in the prison of Rheims[7] in 1839, in the citadel of Ursino and the prisons of Catania[8] in 1822, in 1841 in the hospice for the poor at Naples, where more than 2000 vagabonds were received in consequence of the prohibition of street-begging,[9] in the central prison of Constantinople, where the fever broke out in 1867 whenever it became overcrowded, as it had done on every former occasion,[10] and in 1831 in Hamburg, where all the lodging-houses for tramps were closed on the outbreak of cholera, and the homeless poor, to the number of 293, were packed into a small and dirty space so that typhus fever soon broke out among them, and attacked 119 persons notwithstanding the sanitary regulations that were subsequently adopted.[11] Under the same circumstances, there have been frequent typhus epidemics in the French *bagnes*, for example, in the bagnio of Rochefort in 1839, and in the bagnio of Toulon in 1820-29-33-45-55 and -56.[12] On this point, Barrallier[13] remarks: "All authors are agreed in assigning to crowding the first place among the specific determining causes of typhus. . . . . This cause is present, more than anywhere, in the bagnios, and above all in the floating bagnios, which are old hulks no longer adapted for use in the fleet; in all the epidemics of typhus that have been observed among the convicts, it has always been among those lodged in such places that the disease has taken its rise and has raged with greatest virulence."

Ship typhus.—Allied to the disease that occurs under these circumstances, is the *typhus on board ship*, or the so-called "ship fever;" it used to be a veritable scourge of the marine, but it is hardly ever met with now, thanks to the recent improvements in ventilating apparatus (wind-sails and the like), and, when it does show itself, it is only by reason of

[1] Bischoff, 'Darstellung der Heilungsmethode an der med. Klinik zu Prag,' Prag, 1825, p. 243.
[2] Hoffmann, 'Wiener med. Presse,' 1868, No. 21.
[3] Leiblinger, 'Wiener med. Wochenschrift,' 1868, No. 54.
[4] Cohn, l. c.
[5] Depping, 'Bayr. ärztl. Intelligenzbl.,' 1854, p. 32, and ref. already quoted.
[6] Hellweg, l. c.
[7] Laudouzy, l. c.
[8] Orsini, 'Sulla malatt. febr. svilupp. in Catania,' Catan., 1823.
[9] De Renzi, 'Filiatr., Sebez.,' 1842, Maggio.
[10] Marroin, l. c.
[11] Schmidt, in 'Hamb. Mittheil. a. d. Geb. der Heilkd.,' 1833, ii, p. 243.
[12] See p. 564.
[13] L. c., p. 30.

casual and partly unavoidable misadventures which have led to crowding and insufficient ventilation of the space set apart for the ship's company and the troops on board.

Emergencies of that kind occurred several times within recent date, on board the transports bringing back the French troops from the Crimea, in connexion with which Godelier relates an interesting fact.[1] Two ships sailed at the same time from Kamiesch; one of them arrived at Marseilles without any mishap, having made the passage in thirty days, while the other, which was fifty days on the voyage and was badly ventilated in the lower hold owing to defective apparatus, had an outbreak of typhus during the last thirty days of the passage, in which 30 out of the 800 on board were attacked. The introduction of typhus on more than one occasion in recent times into European ports by Egyptian ships of war is another noteworthy fact. One of these was the case of the frigate Scheah-Gehaed which came to Liverpool in 1861,[2] another the frigate Ibreïmieh, which arrived at Toulon in 1864;[3] in both cases it was a matter of overcrowding in the extremely filthy 'tween decks, which had not been ventilated during the voyage, the hatches having been battened down owing to rough weather. In the first case, the ship's company remained well during the passage [except that many of them suffered from dysentery], and the first cases of typhus were in the pilot and others who had boarded the vessel in the Mersey, [among the attendants at a public bath to which the crew had been sent, and among the patients at the Southern Hospital, into which some twenty of the crew had been admitted for various ailments, but not for typhus].

War-typhus.—The history of *typhus in war* affords further classical proofs of the influence of overcrowding, in filthy and unventilated spaces, upon the development of the fever. This has happened most often, for obvious reasons, in besieged fortresses; but it has occurred not unfrequently among the besieging troops as well, as soon as they have sat down before the place, and the weather has caused the soldiers to pack themselves closely in tents.

Of more recent wars, the Crimean and the Russo-Turkish (1878-79) have obtained a melancholy celebrity in this respect. In the Crimean War, it was principally the English troops that suffered during the winter of 1854-55, their hospital arrangements proving insufficient and the commissariat inadequate for an army that was unusually large for England.

[1] 'Gaz. méd. de Paris,' 1856, p. 470.

[2] Duncan, 'Transact. of the Epidemiol. Soc.,' 1862, i, p. 246.

[3] Gourrier (see p. 564).

On the other hand, in the winter of 1855-56, when the English army was in comfortable quarters and suffered much less from typhus, the disease was very much more intense than the year before, among the French troops who had been placed in a very difficult position when the siege of Sebastopol began, and had to camp on the wet ground in close tents. "There was no typhus in summer," says Jacquot, in his excellent work,[1] "while the soldiers lived in the fresh air, and left their barracks or tents open. With the cold season typhus developed two years in succession; and twice in succession it went away with the return of the warm season, which permitted of ventilation in-doors, and a life in the open air." Again, in the late Franco-German war, France paid her tribute to typhus. According to Michaux and Viry,[2] the disease broke out in Metz at the time of the siege, but it was almost confined to the population of the town, while there was little of it among the troops encamped in the out-works, who felt the scarcity and overcrowding less. The epidemic was of short duration, as Michaux states; for the siege was raised shortly after it broke out, and there was an end to the crowding of the city by people from the surrounding country who had taken refuge in it.

It may be looked upon as a triumph of rational military hygiene in the field, that the German troops during the recent Franco-German campaign, and the Northern troops during the American War of Secession, remained quite free from typhus. In the reports of sickness in the Confederate Army during the Secession War, 1723 cases are put down to "typhus;"[3] but most of these, as the reporter says (p. 213), were cases of typho-malarial fever and typhoid. Accurate information went to show that typhus had occurred only in isolated cases "in connexion with overcrowded and ill-policed camps," and subsequently among such of the troops as had been kept prisoners for a considerable time in the enemy's territory.

Finally, we have to observe that, except in war-typhus in besieged fortresses, where the misery is felt more or less uniformly by all classes of the population, the disease has at

[1] 'Du typhus de l'armée d'Orient,' Paris, 1858, p. 64.

[2] Ll. cc.

[3] 'Report on the Extent and Nature of the Materials available for the Preparation of a Medical and Surgical History of the Rebellion,' Philad., 1865.

all times and everywhere sought out chiefly the crowded and filthy quarters, streets, or houses inhabited by the proletariat, or has even been confined to them.

Cheyne[1] has said with reference to the Dublin epidemic of 1817: "The disease was rare in higher ranks, and there were *very few instances of the fever extending to a second person in any house in which proper attention was paid to cleanliness and ventilation*;" and Murchison[2] writes to the same effect: "In Edinburgh, typhus was confined, even in the worst epidemics, to the crowded parts of the old town occupied by the poor. . . . In the country districts of England, typhus is a rare disease. . . . In London, it is unknown among the middle and upper classes, apart from a very few isolated cases. Drs. Tweedie and Jenner have informed me that they have never seen it among those classes, except in doctors and students." The same characteristic of the disease appears in all the most recently observed local epidemics of typhus, such as at Leeds in 1816 (Hunter), at Whitney in 1818 (Sheppard),[3] at Marburg in 1824 (Rothamel), at Minden in 1848 (Lachmund), at Halle in 1834-44, and -48, at Philadelphia in 1836, at Bonn in 1864 (Moers),[4] at Breslau in 1868-69, at Dorpat in 1866-67 (Behse), at Vienna in 1875 (Oser), and at Stockholm in the same year (Warfwinge). Berlin has never been quite free from typhus since 1867; but the disease has been always strictly confined to those who live in, or frequent, lodging-houses and taverns of the lowest order. Notwithstanding the considerable spread of typhus in Central and Western Germany in 1867-68 in consequence of its introduction by Slavonian hawkers, it never came to be epidemic, and among the better-off classes there was never even a single case of it seen.

Therefore the idea that underlies Hildenbrand's[5] dictum—"the source of all typhus matter is to be looked for solely in concentrated human effluvia"—the idea that *overcrowding in filthy and unventilated rooms affords the essential condition for the development of typhus-foci and for the spread of the disease*, has been completely borne out by the experience of all times. The fact that these social miseries make themselves felt much more severely in the colder seasons and climates than in the warmer months and in tropical or subtropical regions, serves to explain why the largest amount of the sickness falls in winter and spring, and why the lower latitudes enjoy a relative immunity from typhus, if not an absolute one. In like manner, it is perfectly plain that the great progress which sanitation has made in recent times has contributed materially, if not to exterminate the disease, yet to restrict it considerably in many parts of Europe which were often visited by

[1] 'Dubl. Hosp. Reports,' 1818, ii, p. 53.
[2] L. c., p. 58.
[3] 'Edin. Med. and Surg. Journ.,' 1819, July, p. 346.
[4] 'Archiv für klin. Med.,' 1867, ii, p. 36.
[5] 'Ueber den ansteckenden Typhus,' Wien, 1814, p. 374.

severe epidemics of it in former centuries; so that even in these it shows itself only at periods of unusual want, and then only within narrow limits.

§ 139. Origin of Typhus: the Virus.

It is clear that we may have established all the foregoing facts without solving the problem of the *origin of typhus*. The question remains, whether the products of decomposition, or effluvia developing under the circumstances above described—for it is to these that we must look in the last resort—represent *in themselves* the peculiar cause of the disease, or whether the production of the malady requires, besides, a specific *typhus poison* for whose reproduction and potency the decomposition-products and effluvia afford merely an unusually favorable soil. This is the old question that arises in all acute infective diseases; and, so long as we are ignorant of the nature of the morbid poison itself, there is only an *a priori* answer possible. All that can be said for the *autochthonous origin of typhus* out of wretched conditions of living—and has actually been said by Murchison, one of the keenest champions of this doctrine[1]—reduces itself *simply and solely* to the observation that sporadic cases of typhus, or limited epidemic outbreaks, have not unfrequently occurred where it was impossible to trace them to conveyance of a morbid poison from some other place. The evidence is, accordingly, of a negative kind, and even Murchison admits that it is not unassailable. But, on the other hand, it has to be said that we are not entitled to conclude for the spontaneous production of the morbid poison because it has not been possible in every case to find a source whence it had been brought; for, even in infective diseases like smallpox, which can hardly be thought of as arising spontaneously, it is often impossible to track the way by which the disease was conveyed, especially when inanimate objects play a part. Further, it should not be forgotten that all the old hygienic neglect still exists in many parts of the world, without ever becoming the occasion of a

[1] L. c., p. 74.

"spontaneous" outburst of typhus. Lastly, considering the unquestionable power of reproduction in the typhus poison, we shall hardly be able to throw doubt upon its organic nature,[1] and we shall be driven, accordingly, to assume some such specific agent. "I would as soon believe in the spontaneous generation of human beings," says Davies,[2] "as I would in the spontaneous generation of typhus."

As to *the nature of this typhus poison*, inquiries hitherto have not yielded the smallest information.

The discovery of a *typhus fungus* (*Rhizoporus*) made by Hallier[3] has not been confirmed, and it has been pronounced by Rosenstein[4] to be a delusion, on the ground of inquiries which he made on the blood of typhus patients. Obermeier[5] made experiments to infect animals (dogs, rabbits, &c.) with the blood of typhus patients, but with uniform want of success. Neither has any infection followed when a few drops of typhus blood have been introduced, either accidentally or of purpose, under the epidermis of healthy men. The "positive results" which have attended Zuelzer's[6] infection-experiments with animals in a few cases, do not afford the slightest reason for believing that it was typhus which was produced in the animals.

§ 140. Native Habitats.

As to the original habitat of typhus, there is nothing to be said even by way of conjecture. The disease has now, at any rate, become indigenous at many points of the globe, and most decidedly so in those regions of Europe which have been indicated more particularly in the foregoing. Under the influence of the factors which favour its development, it appears as an epidemic there from time to time—without any periodicity in the succession of the outbreaks, as some have thought[7]—and it then spreads not unfrequently over adjoining regions, and sometimes even with a wider sweep, as the war-typhus of former centuries bears witness to on the grand scale.

[1] The organic nature of the virus of typhus had been asserted by Hildenbrand (l. c., p. 141); he compares it to the reproductive germs of animals or plants.

[2] 'Med. Times and Gaz.,' 1867, Oct., p. 429.

[3] 'Virchow's Archiv,' 1868, vol. 43, p. 268.

[4] Ib., p. 419.

[5] 'Centralbl. für die med. Wissensch.,' 1873, No. 36.

[6] 'Viertelj. für die med. Wissensch.,' 1873, No. 36.

[7] 'Edin. Med. and Surg. Journ.,' 848, Oct., p. 374.

§ 141. Contagiousness of Typhus.

There can be no question, on the part of anyone, that typhus counts among the exquisitely *contagious* diseases, or, in other words, that the specific poison of the disease reproduces itself within the sick body, and is eliminated therefrom in a perfect state of potency. At same time the facts adduced above seem to show that unhygienic conditions help the reproduction, or the potency, of that specific agent in some way that we cannot particularise more closely at present, and thereby give occasion to the formation of *typhus centres*. The *diffusion of the disease*, or the conveyance of the morbid poison, takes place equally by the air surrounding the patient, as by personal intercourse and by objects—healthy men or things—which become carriers of the poison that clings to them.

The literature is very rich in examples of the conveyance of typhus by healthy persons, or by articles such as linen or clothes; as well as of the great tenacity with which the poison clings to objects, such as the walls of rooms, furniture, and the like.[1] It has been an often-observed fact, worth noticing in this connexion, that the disease has first broken out among the emigrants from Europe, especially Irish, after they had left the ship on her arrival at an American port, as at Baltimore in 1850.[2] There is some probability in the conjecture that, in these cases, the virus had clung to the effects which they had brought with them. The same conjecture has been put forward, as we have seen, by the French physicians to account for the outbreak of 1868 in the towns of Algiers; and it may apply also to the mode of introduction of typhus into the port of Liverpool by an Egyptian frigate in 1861 (p. 586).

§ 142. Race and Nationality without Influence.

No circumstances of race and nationality give any immunity from typhus.

[1] On the former point, see Zuelzer, l. c., Müller, p. 41, and Murchison, p. 67; on the latter point, the same authorities, and also Becher ('Berl. klin. Wochenschrift,' 1868, p. 502), Behse, Maclagan, and Davies.

[2] Wynne, 'Amer. Journ. of Med. Sc.,' 1852, April, p. 417.

In the epidemic observed by Brown[1] in the poor-house at Boston, and in that by Klapp[2] in the poor-house at Philadelphia, as many negroes as whites sickened. In the Philadelphia epidemic of 1836, the number of cases in negroes and mulattoes was considerably greater than among whites (Gerhard); and Murchison[3] says that he has not unfrequently seen Indians and Africans with typhus in the London Fever Hospital.

It cannot be said of typhus, as it can of typhoid, that there is any *acclimatisation*, or, in other words, a diminished or increased susceptibility to the morbid poison by long residence in localities or tenements that are often infected, or that constitute typhus-centres. It is only having survived the disease once that affords a degree of protection from the morbid poison of typhus, as in the acute exanthemata, although the protection is feebler in degree than in the case of the latter fevers.

[1] 'New England Journ. of Med.,' 1818, vii, p. 105.
[2] 'Amer. Med. Recorder,' 1821, iv, p. 80.
[3] L. c., p. 49.

CHAPTER XII.

RELAPSING FEVER AND BILIOUS TYPHOID.

§ 143. The Scotch and Irish Epidemics; Present Geographical Distribution.

The history of relapsing fever and of bilious typhoid, which I take to be special modifications of one and the same morbid process,[1] does not permit of being followed back beyond the eighteenth century, so far as epidemiography is our guide; although it is probable that the disease in both its forms had occurred before, and had been confounded with other diseases allied to it in symptoms, especially with malarial fever and other so-called typhous fevers.[2]

[1] It is well known that Griesinger has the merit of having first correctly recognised the peculiar features of bilious typhoid, and of having given a detailed account of the disease from the observations that he made in Egypt; he pointed out the difference between it and bilious remittent and yellow fever on the one hand, and on the other hand between it and relapsing fever, which had first become accurately known since 1842 through the Scottish physicians; and he showed that both these forms of disease were to be regarded as modifications of one morbid process. Lebert and others subsequently raised objections to the identification of relapsing fever and bilious typhoid, but, as I think, without reason. I lay no stress on the fact that the two forms occur very often together in the same epidemic; for that is true also of relapsing fever and typhus, which are, without doubt, quite different processes. The decisive facts in my opinion are: that there are clearly marked transition-forms between relapsing fever and bilious typhoid, which, from the point of view of symptoms and morbid anatomy, incline sometimes to the character of the one and sometimes to that of the other; and above all, that the same morbid poison underlies both forms, as we shall see later on.

[2] I have searched in vain in the descriptions which the physicians of the sixteenth and seventeenth centuries have given of the fever epidemics observed by them, for any indications of relapsing fever that would be in some measure precise. There is certainly mention made in them, and still more frequently in the eighteenth-century accounts of "bilious," "mucous," and "putrid" fevers, of "relapses" which the patients had suffered; but it is clear that the reference

The first reliable information as to the occurrence of relapsing fever on *European* soil is found in the records of pestilence in *Ireland* and *Scotland* about the beginning of the eighteenth century. Rutty speaks of an epidemic at Dublin in 1739 in these terms :[1]

"It [the disease] terminated sometimes in four, for the most part in five or six days, sometimes in nine, and commonly in critical sweat. . . . . The crisis, however, was very imperfect, for they were subject to relapses, even sometimes to a third time."

Subsequent notices of epidemics observed there in 1745, 1748, and 1764-65 are to the same effect; and it is from that period (1741) that we have the earliest accounts of relapsing fever in Scotland.[2] The same coincidence of the epidemics in Ireland and Scotland had been observed also during the first thirty years of the present century, in 1799—1800,[3] 1817-19,[4] and 1826-27.[5] In the last of these the disease often assumed the character of bilious typhoid, so much so that Graves and O'Brien were constrained to explain cases of that kind as yellow fever. In the summer of 1842 relapsing

is not to relapses but to incidence of the disease afresh (in typhus or typhoid). Spittal's view ('Edin. Monthly Journ. of Med. Sc.,' 1844, iv, p. 177), that some of the fevers described by Hippocrates (in 'Epidem.,' lib. i, sect. i, § 3, sect. ii, § 4, sect. iii, § 9, ed. Littré, ii, p. 612, 626, 660), are to be taken as relapsing fever, appears to me to be quite erroneous; it is clear that Hippocrates speaks there of bilious remittent malarial fever.

[1] 'Chronological History of the . . . prevailing diseases in Dublin,' Dubl., 1770, p. 90.

[2] Stark, in 'Transact. of the Epidemiol. Soc.,' 1867, ii, p. 309.

[3] In the account by Barker and Cheyne of the typhus epidemic of 1817–19 in Ireland ('Account of the Fever lately Epidemical in Ireland,' i, p. 20) it is stated: "Certain it is, that the fever in 1800 and 1801 very generally terminated on the 5th or 7th day by perspiration; that the disease was then very liable to recur." Stark (l. c.) gives an account of the epidemic in Scotland in those years.

[4] For the Irish epidemic, see Harty, 'Historical Account of the Contagious Fever,' &c., Dublin, 1820, p. 131, App. viii; Rogan, 'Observations on the Condition of the Middle and Lower Classes in the North of Ireland, &c.,' London, 1819, p. 27; Barker and Cheyne, l. c., i, p. 211. For the Scottish epidemic, see Stark, l. c.; Duncan, 'Report of the Practice in the Clinical Wards of the Infirmary of Edinburgh,' 1817–18,' Edinb., 1818; Welsh, 'On the Efficacy of Bloodletting in the Epidemic Fever of Edinburgh,' Edinb., 1819, p. 16.

[5] See (for Ireland) O'Brien, 'Transact. of the College of Phys. in Ireland,' v, 1828; Reid, ib.; Graves, 'Clinical Reports,' part i, p. 53, *seq.*; Stark, l. c., and the review of Burne's 'Treatise on the Typhous Fever (Lond., 1828), in the 'Edinb. Med. and Surg. Journ.,' 1828, Octr., p. 413 (for Scotland).

fever broke out anew in several parts of Ireland, and continued to be epidemic there to a greater or less extent until 1848.[1] In Scotland it had already appeared in the County of Fife in the summer of 1841,[2] chiefly in the form of bilious typhoid, and it reached a more general diffusion first in 1842-44 in several of the larger towns of the country, such as Glasgow,[3] Edinburgh,[4] Leith,[5] Dundee,[6] and Aberdeen.[7] This was, indeed, the epidemic that first made the medical world more accurately acquainted with the peculiar nature of relapsing fever. It appears from Cormack's[8] statements that in Edinburgh also the disease often bore the character of bilious typhoid. Fresh outbreaks of relapsing fever took place in Ireland and Scotland in 1847-48,[9] in which years the disease showed itself also in London,[10] Croydon,[11] Liverpool, Manchester,[12] and other towns of *England*. Another series of outbreaks occurred in 1868-73. There are no particulars of that epidemic for Ireland; it visited Edinburgh,[13] Glasgow,[14] and other towns in Scotland in 1869; while in London it had already begun in the latter

[1] Lalor, 'Dublin Journ. of Med. Sc.,' 1848, Feb.

[2] Goodsir, 'Edin. Med. and Surg. Journ.,' 1845, Jan., p. 134.

[3] Reid, 'Lond. Med. Gaz.,' 1843, Dec.; Mackenzie, ib., Nov., and in 'Edin. Monthly Journ.,' 1844, Feb.; Smith, 'Edin. Med. and Surg. Journ.,' 1844, Jan., p. 67, July, p. 62; Perry, ib., July, p. 81; Orr, ib., 1845, April, p. 387.

[4] Cormack, 'Natural History of the Epidemic Fever, &c.,' Lond., 1843; Craigie, 'Edin. Med. and Surg. Journ.,' 1843, Oct., p. 410; Henderson, ib., 1844, Jan., p. 201.

[5] Jackson, ib., 1844, April, p. 417.

[6] Arrott, 'Scot. and North of Engl. Med. Gaz.,' 1843, Dec., p. 133.

[7] Kilgour, 'Brit. and For. Med.-Chir. Review,' 1844, July.

[8] L. c., p. 23.

[9] For Ireland: see Donovan, 'Dubl. Med. Press,' 1848, p. 67; and notices in the 'Dubl. Quart. Journ. of Med. Sc.,' 1848, vii, pp. 64, 340, viii, pp. 1, 270. For Scotland: Account in 'Edin. Monthly Journ.,' 1847, July, p. 71, and Robertson, ib., 1848, Dec., p. 368 (Edinburgh); Paterson, 'Edin. Med. and Surg. Journ.,' 1848, Oct., p. 371 (Leith); Orr, ib., April, p. 363, and Steele, ib., July, p. 145 (Glasgow).

[10] Hughes, 'Lond. Med. Gaz.,' 1847, Nov., p. 923; Jenner, 'On the Identity or Non-identity of Typhus and Typhoid Fevers.' (See *Note* [5], p. 621.)

[11] Bottomley, 'Prov. Med. and Surg. Journ.,' 1847, Dec., p. 701.

[12] Duncan, ib., Nov., p. 524.

[13] Muirhead, 'Edin. Med. Journ.,' 1870, July, p. 1.

[14] Notices in 'Brit. Med. Journ.,' 1870, Sept., p. 341, Oct., p. 397, Dec., p. 610 Tennent, 'Glasgow Med. Journ.,' 1871, May, p. 354.

half of 1868, chiefly in a quarter inhabited by Irish and by poor Jewish emigrants from Poland.[1] It was observed also in the autumn and winter of 1868 in North Shields, where it took the form of bilious typhoid in many cases,[2] as well as in Monmouthshire;[3] but in these localities as well as in Liverpool,[4] Leeds,[5] Manchester,[6] and other places, its more considerable epidemic prevalence was in 1869 and 1870. Of its persistence into 1873 there are accounts from London, the County of Kent,[7] and Newcastle.[8]

The first ascertainable occurrence of relapsing fever in other parts of Europe falls much later than its appearance in the British Islands. The earliest notices of it come from Russia, from Odessa[9] in 1833, and from Moscow, where there was a severe epidemic of bilious typhoid in the winter of 1840-41.[10] The disease in either form did not become at all general in Russia until 1863. In the autumn of that year relapsing fever reappeared in Odessa,[11] in the summer of the year following there was a widespread epidemic in the Governments of St. Petersburg,[12] Novgorod,[13] Moscow,[14] and others, in 1865 it appeared in Livonia[15] and Finland,[16] in 1866

[1] Weber, 'Lancet,' 1869, Feb., pp. 221, 255; Murchison, ib., Oct., p. 503, Nov., p. 647.

[2] Scott, ib., Dec., 1868, p. 796.

[3] Notice, ib., 1870, Nov., p. 684.

[4] Gee, 'Brit. Med. Journ.,' 1870, Sept., p. 246.

[5] Robinson, 'Lancet,' 1871, May, p. 644.

[6] Notice in 'Brit. Med. Journ.,' 1870, Oct., p. 466.

[7] Notice in 'Lancet,' 1872, Jan., pp. 29, 48.

[8] Armstrong, ib., 1873, Jan., p. 48.

[9] Bernstein, 'Gaz. méd. de Paris,' 1865, p. 426.

[10] Excellent accounts of this epidemic have been given by Heimann ('Hufeland's Journ.,' xcvi, pt. 3, p. 94), by Pelikan ('Mosk. Mitth. a. d. Geb. der Hlkd.,' Leipz., 1845, p. 111), and by Levestamm (ib., p. 2).

[11] Bernstein, l. c.

[12] Botkin, 'Berl. klin. Woch.,' 1864, p. 513; Herrmann and Küttner, 'Die febris recurrens in St. Petersburg,' Erlang., 1865; Zorn, 'Petersb. med. Zeitschr.,' 1865, ix, p. 1; Kernig, ib., 1867, xii, p. 177; Herrmann, ib., 1867, p. 1, and 1870, xv, p. 385. According to the last of these accounts, the disease had been epidemic in St. Petersburg almost continuously from 1863 to 1868.

[13] Donbowitzky, 'Gaz. des hôpit.,' 1865, Avril.

[14] Sacharjin, 'Wiener med. Wochenschr.,' 1866, p. 841.

[15] Girgensohn, 'Arch. für klin. Med.,' 1877, xix, pp. 19, 246; Behse, 'Petersb. med. Zeitschr.,' 1868, xiv, p. 1.

[16] Edholm, 'Hygiea,' 1868, Förhdl., p. 73; Palmberg, ib., 1870, p. 68; Holst, 'Nord. med. Arkiv,' 1879, xi, No. 8, p. 18.

in Siberia,[1] and in 1868 in Poland.[2] The epidemiographical information from that country for these and following years is too scanty to imply more than that the sickness in the period subsequent to this was widely prevalent over the whole of Russia, that many places were visited by it repeatedly, and that it has been observed as late as the winter of 1878-79 among the Russian troops in Bulgaria.[3] The valuable papers by Zorn (l. c.) and Moschutkowsky,[4] founded on their observations made respectively at St. Petersburg in 1863-64 and Odessa in 1873-76, speak of the disease assuming the type of severe bilious typhoid.

In the *Scandinavian* kingdoms relapsing fever seems to have been observed to a very slight extent hitherto. From *Norway* there is only one notice of its proper epidemic prevalence; in the district of Vadsöe, inhabited by a poor fishing population, more or less numerous cases of relapsing fever had occurred every year from the autumn of 1851 to the spring of 1861,[5] and the disease reappeared in 1865, having been imported on that occasion, it was said, from Finland.[6] From *Denmark* there is no information whatsoever as to the occurrence of relapsing fever, and for *Sweden* there are merely references to isolated cases in various parts of the kingdom in 1874 and 1875.[7]

Next to the British Islands and Russia, Germany has been hitherto the country most severely visited by relapsing fever. The first notices of it there, relating, however, only to isolated cases, come from the severe typhus epidemics of 1847 and 1848 in Upper Silesia,[8] and in Königsberg, from which we have an account by Lange of several cases of bilious typhoid observed by him.[9] It was in 1868 that the disease first broke out over a wide area and in true epidemic diffusion,

[1] Lewonewsky, quoted by Rudnew in 'Virchow-Hirsch's Jahresber.,' 1867, ii, p. 267.

[2] According to Wyss and Bock, 'Studien über Febris recurrens,' Berl., 1869, p. 10.

[3] Maximowitsch, 'Petersb. med. Woch.,' 1879, Nos. 6, 7.

[4] See p. 613.

[5] Danchertson, 'Norsk. Mag. for Laegevidensk,' 1865, xix, p. 76.

[6] Boeck, ib., 1874, iii, R. iv, p. 241.

[7] 'Sundhets-Kolleg. Berättelse för år 1874,' p. 7, 1875, p. 16.

[8] See Dümmler, l. c., p. 336; Deutsch, l. c.; Bärensprung, l. c., p. 481.

[9] 'Beobacht. am Krankenbette,' Königsberg, 1850, p. 285.

and it was undoubtedly a consequence of importation from Poland or Russia. In the earlier half of that year relapsing fever showed itself almost simultaneously in Pomerania (Greifswald)[1] and Upper Silesia,[2] then at Königsberg and other places in East Prussia,[3] at Kulm, Marienwerder, and other places in West Prussia,[4] and further at Posen,[5] Breslau,[6] and Stettin.[7] The disease was imported either from the Province of Prussia or from Silesia in the autumn of 1868 into Magdeburg[8] and Berlin,[9] where it lasted through the winter, as it did also in Greifswald and Breslau. At many places the outbreak was confined to isolated cases, and, except in Breslau where the cases numbered 476, it nowhere reached to the dimensions of a considerable epidemic. There was a second visitation in 1871-72 at Greifswald,[10] Posen,[11] Stettin,[12] Berlin,[13] and Breslau;[14] but this time also its extent was but small, Breslau being again the exception, with 466 cases. The third, and hitherto the last outbreak, in 1878-79, was of the same kind; more or less numerous cases of relapsing fever were observed in Dresden,[15] Berlin,[16]

[1] Mosler, 'Corrspdzbl. mittelrhein. Aerzte,' 1868, ii, p. 157, and 'Berl. klin. Wochenschr.,' 1869, No. 31; Brodziak, 'Das Vorkommen des Typhus recurrens im Jahre 1868–69 in Greifswald,' Dissert. Greifsw., 1874.

[2] Wyss und Bock, l. c., p. 10.

[3] Bernhardt, 'Berl. klin. Woch.,' 1869, No. 2.

[4] Wyss, l. c., p. 9.

[5] Hirschberg, 'Berl. klin. Woch.,' 1868, p. 359; Svidersky, 'Deutsche Klinik,' 1868, p. 467.

[6] Wyss und Bock, l. c.; Pastau, in 'Virchow's Archiv,' 1869, vol. 47, p. 161; Graetzer, 'Ueber die öffentl. Armenkrankenpflege und die Febris recurrens Breslaus im J. 1868,' Bresl., 1869, p. 29; Lebert, 'Arch. f. klin. med.,' 1870, vii, p. 461.

[7] Steffen, 'Jahrb. für Kinderhlkd.,' 1869, ii, p. 61.

[8] Aufrecht, 'Berl. klin. Woch.,' 1869, Nos. 29, 30.

[9] Riess, ib., 1868, p. 229, 1869, p. 327; Obermeier, in 'Virchow's Archiv,' 1869, vol. 47, p. 161. In Leipzig also a few imported cases occurred (Wunderlich, Archiv der Hlkd.,' 1869, p. 314).

[10] Treibel, 'Febris intermittens und febr. recurrens,' Dissert. Greifsw., 1872.

[11] Kaczorowsky, 'Berl. klin. Woch.,' 1872, No. 23.

[12] Pilz, 'Jahrb. für Kinderhlkd.,' 1872, vi, p. 66.

[13] Semon, 'Zur Recurrens-Epidemie in Berlin 1871–72,' Dissert., Berlin, 1873; Budberg, 'Ueber febris recurrens, &c.,' Dissert., Berlin, 1873.

[14] Litten, 'Archiv für klin. Med.,' 1874, xiii, pp. 125, 281.

[15] Müllendorff, 'Deutsche med. Woch.,' 1879, Nos. 48—50.

[16] Riess, ib., Nos. 51, 52; Winzer, 'Beobacht. über febris recurrens,' Dissert., Berlin, 1880.

Swinemünde,[1] Danzig,[2] Greifswald,[3] Halle,[4] Stralsund,[5] Brunswick,[6] Magdeburg,[7] Breslau,[8] and other places in North Germany. The bilious-typhoid form has been much rarer here than in Great Britain or in Russia; isolated cases of that type have been reported by Kaczorowsky from Posen in 1872, and by v. Meurers[9] from the Berlin Charité the same year.

There are no accounts of relapsing fever from South Germany, with the exception of a notice of an epidemic at Giessen in 1878-79.[10] On the other hand, there are such from Austria, as in 1847 from Cracow[11] and other parts of Galicia,[12] where the disease appears to have assumed for the most part the type of bilious typhoid. It reappeared there as an epidemic in 1865-67, in the county prisons at Tarnopol,[13] Belz, Gross-Mosty, and other places,[14] and about the same time at Prague.[15] Isolated cases showed themselves at Cracow in 1875, and the disease again attained a considerable epidemic diffusion there in 1877-78.[16]

Excepting *Belgium*, where a few cases were seen in 1867[17] (at Brussels, Bruges, Blankenberghe, and other places), the *West and South-West of Europe* appear to have been hitherto exempt, that is to say, *Switzerland*, *France*, *Italy*, and the *Iberian Peninsula*. On the other hand, there are numerous, although not always reliable, data, some of them even from earlier times, pointing to the somewhat frequent occurrence of relapsing fever in the *Levantine States* and *Islands*

[1] Caspar, 'Berl. klin. Woch.,' 1880, Juni, p. 329.
[2] Knipping, 'Arch. für klin. Med.,' 1880, xxvi, p. 10.
[3] Mosler, 'Deutsche med. Woch.,' 1879, No. 11; Kühn, ib., 1880, No. 23.
[4] Risel, ib., No. 11.
[5] Hecht, ib., No. 14.
[6] Enke, 'Ueber den Rückfalltyphus, &c.,' Magdeb., 1879. [7] Ib.
[8] Spitz, 'Arch. für klin. Med.,' 1880, xxvi, p. 139.
[9] 'Die hämorrhagische Diathese und ihr Vorkommen bei Recurrens,' Dissert., Berlin, 1873,
[10] Lachmann, 'Archiv für klin. Med.,' 1880, xxvii, p. 526.
[11] Warschauer, 'Allg. Wiener med. Ztg.,' 1878, No. 44.
[12] Prchal, 'Oest. med. Woch.,' 1874, Nos. 49, 51.
[13] Leiblinger, 'Wien. med. Woch.,' 1868, Nos. 54—55.
[14] Account in 'Allgem. militär-ärztl. Ztg.,' 1866, p. 93.
[15] Pribram und Robitschek, 'Prager Viertelj. für pract. Heilk.,' 1869, ii, p. 108.
[16] Warschauer, l. c.
[17] V. Biervliet, 'Bull. de l'Acad. de méd. de Belgique,' 1867, p. 843.

of the Mediterranean, whether belonging to Europe, Asia, or Africa.

Relation to bilious typhoid.—Our earliest mention of a kind of sickness in those regions with the character of bilious typhoid, dates from the second or third decade of this century, and it is found in observations by the surgeons of the English army and navy relating to Malta, the Ionian Islands, and the crews of English ships of war. The most detailed account is that of Burnett,[1] who clearly confounds, however, bilious typhoid and severe remittent malarial fever. There are more precise statements by Denmark[2] and Cutbush,[3] both of whom give a sketch of bilious typhoid which cannot be mistaken; and they agree in saying that the disease has nothing in common with malarial fever, being distinguished from the latter by its communicability or contagiousness. Bonnar[4] also, in describing the Irish typhus (*i.e.* relapsing fever) of 1817, remarks that the epidemic fever which he had seen in the Mediterranean in 1810-12, differed from the former only in the shortness of its course and in the bilious symptoms. It is possibly to this class also, that the "typhus epidemics" belong, which occurred in 1817 at Spalato and other places in Dalmatia,[5] and in 1835 at Athens,[6] as well as the "bilious typhus" which Rigler saw at Constantinople in 1843.[7] We find another interesting addition to the history of the prevalent relapsing fever or bilious typhoid of those parts, in the so-called "Cyprus fever" with which the English profession has become more intimately acquainted since the recent occupation of the island by the British Government. In an account dating from Cyprus,[8] there is a description given of the disease which leaves no doubt that we have here to do with a form of relapsing fever running its course with bilious symptoms; and in a subsequent notice,[9] in which, however, the disease is evidently confounded with malarial fever, it is stated that the same kind of fever is known in the East under the names of "Levant fever," "Bukowina fever," "Smyrna fever," and the like. We possess a

[1] 'Practical Account of the Mediterranean Fever,' &c., Lond., 1816.

[2] 'Med.-Chir. Transact.,' 1815, vi, p. 296, from observations at Port Mahon, Minorca.

[3] 'Amer. Med. and Philos. Register,' 1811, i, p. 356. It is a very striking circumstance that Cutbush, who calls the disease "typhus," draws attention to the fact that severe inflammations of the eye occurred not unfrequently during convalescence—an incident which is well known to be very characteristic of relapsing fever.

[4] 'Statement of the Results of Practice in the Continued Fevers,' Lond., 1818, p. 20.

[5] Frari, 'Storia della febbre epid. che regnò a Spalato nell' anno 1817,' Padova, 1818 (Ozanam, iii, p. 203).

[6] Rothlauf, 'Die Epidemie in Athen im Sommer 1835,' Athen, 1836.

[7] 'Die Türkei, &c.,' ii, p. 399.

[8] 'Brit. Med. Journ.,' 1878, Oct., p. 574.

[9] 'Lancet,' 1878, Dec., p. 819.

description of this Bukowina fever by Engel,[1] who makes it out to be a contagious typhus prevalent every year in the winter season, and almost exclusively among the poorest people; and in his description he draws a picture, which can hardly be mistaken, of relapsing fever running a course with bilious symptoms, or of typical bilious typhoid. For the Smyrna fever, also, there is an earlier notice by Aubert,[2] who likewise describes it as a peculiar kind of typhus complicated with jaundice ("une espèce particulière de typhus avec teinte jaune")—probably the same disease which Röser[3] had seen there, and described under the name of "yellow fever."

The last points in the distribution-area of relapsing fever and bilious typhoid are found in the *North African Coast* territories. In Pruner's[4] account of the diseases of *Egypt*, there are some indications of bilious typhoid; after him Veit[5] gave a good description of the disease from his observations at Cairo in 1836; then comes the original and suggestive work of Griesinger,[6] who was the first to give a complete explanation of this disease, and of its relation to relapsing fever and the so-called "typhous fevers." Also in *Nubia*, the bilious typhoid appears to be endemic; the earlier statements of Russegger[7] on a "malignant-bilious typhous fever" prevalent there, which did not intermit in its course and which spread by contagion, have been confirmed in the recent accounts by Hartmann[8] of the occurrence of relapsing fever and bilious typhoid in those regions. The accounts from India, which we shall come to next, make mention of the disease in *Abyssinia*. Finally, Arnould[9] reports from *Algiers* that in the typhus epidemic of 1867 in Constantine, there were also many cases of relapsing fever observed.

One of the most extensive centres of relapsing fever and bilious typhoid is met with in *India*. In the first edition of this work (1860), I had thrown out the conjecture that

[1] 'Oest. med. Jahrb.,' 1847, iii, p. 249.

[2] 'De la peste, &c.,' Paris, 1840, p. 10.

[3] 'Ueber einige Krankheiten des Orients,' Augsb., 1837, p. 31.

[4] 'Die Krankheiten des Orients,' p. 381.

[5] 'Württemb. med. Correspdzbl.,' 1851, No. 10, p. 313.

[6] 'Archiv für physiol. Hlkd.,' 1853, xii, p. 29, and in 'Virchow's Handb. der spec. Pathol.'

[7] 'Reisen in Europa, &c.,' iii.

[8] 'Naturgeschichtlich-med. Skizze der Nilländer,' Berl., 1865.

[9] 'Arch. gén. de méd.,' 1867, i, p. 695, ii, p. 50.

various forms, corresponding to the "typhous fevers" of German authors, were concealed behind the "remittent and continued fevers" of Anglo-Indian physicians, and that relapsing fever and bilious typhoid were probably among them. This conjecture has been borne out by the most recent facts reported from India; during the last thirty years numerous accounts of epidemics of relapsing fever and bilious typhoid have come in from various parts of the country, especially from Bengal, the North-West Provinces, and the Punjaub; and some of the writers state that these diseases had often been seen in India before, but had been confounded with other forms of fever, or not recognised by their distinctive characters.[1]

The first definite information as to its occurrence in India is found in Sutherland's[2] account of an epidemic which was prevalent in Patna from December, 1856, to May, 1857, and was probably spread over a still larger part of Lower Bengal. Next, in June, 1859, relapsing fever and bilious typhoid broke out in Sangur, as a disease quite unknown, it is said,[3] to the then resident physicians; and it travelled thence in a north-westerly direction towards the Ganges, spreading widely over the districts of the North-West Provinces situated between the Ganges and Jumna, and over the territories of Behar and Benares.[4] It deve-

[1] Lyons, for example, in his sketch of the history of relapsing fever in India ('Treatise on Relapsing or Famine Fever,' Lond., 1872), takes this ground; but he has quite clearly fallen into the opposite error, inasmuch as he has mixed up altogether alien things, even epidemics of dengue and malarial relapses, with febris recurrens, and has ignored the occurrence of bilious-remittent malarial fevers, taking them to be bilious modifications of relapsing fever. He has thus introduced a new source of confusion into the history of pathology in India. The same error underlies the account by Chevers of the history of relapsing fever in India, published in the 'Med. Times and Gaz.,' 1880, Jan., p. 115, Feb., p. 145.

[2] 'Indian Annals of Med. Sc.,' 1859, Jan., p. 52. In describing the disease, he says: "In nearly all the cases, the fever abated on the 5th or 6th day, but a relapse almost invariably took place after an interval of two or three days' duration." The disease sometimes assumed the type of simple relapsing fever, or the same complicated with bilious symptoms, sometimes of a pronounced bilious typhoid, to which the morbid anatomy (especially the condition of the spleen) was found to correspond completely.

[3] 'Madras Quart. Journ. of Med. Sc.,' 1862, April, p. 423.

[4] Clark, 'Reports upon Epidemic Fever of a Contagious type, in some of the Jails in the N.-W. Provinces,' 1861 (based on observations in the districts of Agra, Meerut, Allahabad, Benares, and Ghazipoor). Walker ('Edin. Med. Journ.,' 1861, May, p. 986) takes the disease for typhus, on the ground of his observations in the Central Prison at Agra.

loped often into an epidemic, especially in prisons, where it mostly assumed the type of severe bilious typhoid. The same is true of its prevalence in 1863-66 at many places in the Punjaub, where it subsequently spread even to the civil and military residents.[1] In 1868 it appeared in India afresh, and under circumstances which pointed to importation from Abyssinia. The fever occurred among a detachment of mule-drivers who had been sent the year before from the Punjaub to Abyssinia to the number of 5000, and were re-embarked for home at the close of the campaign in the autumn of 1868; it broke out after they landed at Bombay, and communicated itself to the people on board the craft which took them up the Indus, continuing to spread, on their arrival in the Punjaub, over the districts of Lahore, Rawul Pindee, Amritsar, and Mooltan. During the passage from India to Abyssinia they had enjoyed perfect health, as well as during the first part of the campaign; they afterwards suffered from diarrhœa, dysentery, scurvy, and "febris remittens," which probably means that they were already suffering from relapsing fever.[2] The first reliable information as to the occurrence of relapsing fever and bilious typhoid in Bengal dates from 1863-65, and relates to the epidemic diffusion of the disease in the districts of Burdwan and Nuddea,[3] as well as among the coolies shipped from Calcutta to the colonies (hence the name of "coolie fever"[4]), its appearance in *Mauritius* and *Réunion* being connected with that importation.[5] In the latter also the disease often assumed the type of bilious typhoid, so that, as Azéma remarks, practitioners believed that it was yellow fever which they had before them. In the Bombay Presidency it showed itself first, so far as we may judge from available facts, in 1864 in the city of Bombay,[6] the year after in Malwa and Gujerât,[7] and at the same time in the Deccan, whence we have information[8] from Bangalore. Since 1866 relapsing fever and bilious typhoid would appear to have spared scarcely a single territory of Hindostan;[9] and Lyons saw it also in *Lower India*[10] in 1871 at Kasalong

[1] See Gray, 'Lancet,' 1869, Nov., p. 648; Smith, 'Ind. Med. Gaz.,' 1867, May (for Rawul Pindee); Bateson, 'Ind. Annals of Med. Sc.,' 1867, April (for the prisons of Kurnaul and Umballa); Ross, 'General Report on the Lunatic Asylums . . . . in the Bengal Presidency,' 1868 (for an epidemic of relapsing fever in the Lahore Lunatic Asylum).

[2] Gray, l. c.

[3] Lowe, 'Madras Quart. Journ. of Med. Sc.,' 1866, July, p. 101.

[4] Smith, 'First Report of the Sanitary Commissioner for Bengal,' 1868, p. 534; notice in 'Ind. Med. Times and Gaz.,' 1867, Dec.

[5] Azéma, 'Union méd.,' 1866, Août, p. 338; Bouvet, 'Arch. de méd. nav., 1867, Oct., p. 286; McAuliff, ib., 1868, Févr., p. 97; Sillian, 'Fièvre à rechutes, Paris, 1869.

[6] Carter, 'Med. Times and Gaz.,' 1878, June, p. 634, and in 'Med.-Chir. Transact.,' 1878, lxi, p. 273.

[7] Brodrick, 'Madras Quart. Journ. of Med. Sc.,' 1866, July, p. 229.

[8] Sutherland, ib., 1866, April, p. 285; Lowe, ib., l. c.

[9] See Chevers, l. c.

[10] 'Ind. Annals of Med. Sc.,' 1872, July, p. 13.

in the hills above Chittagong. Its most considerable prevalence was in 1876-77, when it took largely the form of bilious typhoid, in many parts of India, particularly the Deccan and the Bombay Presidency.[1]

The first and only mention of the disease in *China* dates from the same period in which relapsing fever and bilious typhoid attained their more considerable diffusion on Indian soil, or from 1864-65; Morache[2] states that during the typhus epidemic of 1864-65 at Pekin and other places in Northern China relapsing fever was also epidemic; and Murray[3] mentions a destructive disease which broke out first in 1865 in a prison at Hong Kong, whence it spread generally among the native inhabitants of the town. The disease ran its course with symptoms like those of yellow fever, but with more rapid development of the typhous condition, and it showed itself to be highly contagious. I believe that I shall not go far wrong if I recognise in this sickness an epidemic of bilious typhoid.

Australia and *Polynesia* hitherto (or at least up to 1875, the date of a paper by Bourse[4]) have been quite exempt from relapsing fever and bilious typhoid. On the other hand, it has found its way to *North America* from England or Ireland, but has been hitherto confined[5] to a few of the *Eastern States of the Union*, and has not attained great dimensions as an epidemic. It showed itself first in 1844 at Philadelphia among emigrants who had arrived from Liverpool, there being a few cases also among those in charge of them;[6] in 1847 it broke out in New York under the same circumstances, and it is said to have spread, in that and the year following, over some of the adjoining States.[7] In 1850 Flint[8] saw a few cases of relapsing fever at Buffalo, among

[1] Carter, ll. cc.; Hunter, 'Med. Times and Gaz.,' 1877, Nov., p. 569; notice in 'Lancet,' 1877, July, p. 92.

[2] 'Rec. de mém. de méd. milit.,' 1866, Févr., p. 142; and in 'Annal. d'Hyg.,' 1870, Janv.

[3] 'Lancet,' 1866, June, p. 638.

[4] 'Arch. de méd. nav.,' 1876, Juin.

[5] The account by Donbowitzky ('Gaz. des hôpit.,' 1865) of the prevalence of relapsing fever in 1857–58 at Sitka (New Archangel, in Alaska), I do not consider to be altogether trustworthy.

[6] Clymer, 'New York Med. Record.,' 1870, Feb., p. 575.

[7] Dubois, 'Transact. of the Amer. Med. Assoc.,' 1849, i, p. 382.

[8] 'New York Med. Journ.,' 1870, March.

imported cases of typhus. In 1869 the disease was again imported into Philadelphia;[1] at first it remained almost limited to immigrants, but in the two following years it began to spread, although to a moderate extent, to several other places in Pennsylvania.[2] About the same time there was a fresh outbreak in New York,[3] where it was again among the Irish that the first cases occurred; but on this occasion there seems to have been no extension to other parts of the State.

Up to the present time nothing is known of the occurrence of relapsing fever or bilious typhoid in *Central* and *South America*.

§ 144. Coincidence with Typhus.

If the sketch here given of the history of relapsing fever (or bilious typhoid) falls unquestionably far short of the reality, if the disease both in past centuries and in later and most recent times has occurred much more frequently and over a much wider area than we might conclude from the data before us, still those data point to a much more limited *area of distribution for relapsing fever* than for typhus. This fact will appear all the more striking, and all the more noteworthy for the pathogenesis of both diseases, when we reflect not only that *relapsing fever and typhus coincide* remarkably often in time and place (relapsing fever appearing to be associated in a very conspicuous manner with epidemics of typhus), but also when we consider that there is an almost complete agreement in their relation to those external influences which, as etiological factors, appear more or less to further their development.

This *coincidence of relapsing fever and typhus in time and place*, which showed itself in the first authenticated epidemics of the former disease on Irish soil, has assumed various forms. Frequently the two diseases have occurred simul-

[1] Parry, 'Amer. Journ. of Med. Sc.,' 1870, Oct., p. 336; Jacquet, 'Philad. med. and Surg. Rep.,' 1870, June, p. 469.

[2] 'Transact. of the State Med. Soc. of Pennsylvania,' 1871 and 1872.

[3] Flint, l. c.; Harris, 'First Annual Report of the Board of Health of the City of New York,' 1871; Clark, 'New York Med. Record,' 1870, March 1, 15; Loomis, ib., p. 5.

taneously, in sporadic cases, or in epidemic diffusion, as in 1800—1801 and 1817—1819 at many places in Ireland, and in 1826 at Dublin; in 1847-48 at Edinburgh, Leith, Glasgow, and other places in Scotland, in 1847 at Cracow, in 1865-69 in the Baltic Provinces of Russia, in 1865 at Pekin, and in 1876 in Constantine. Or there have been only a few cases of relapsing fever during the epidemic prevalence of typhus, as in 1847 and 1867[1] in Upper Silesia, and in 1853-54 among the troops in the Crimean war. Or, again,—and this appears to have been the most frequent case—relapsing fever has first occurred as an epidemic, and typhus has assumed an epidemic character towards the end of it, as at St. Petersburg in 1865, in Monmouth and other parts of England in 1868-69, and at Berlin in 1871. Or, finally, the typhus epidemic has preceded the relapsing fever, so that the cases of relapsing fever have begun to be common on the subsidence of the former, as at Berlin in 1868, at Liverpool and Glasgow in 1870, and at Posen in 1872.

§ 145. Not Dependent on Climate.

Relapsing fever in all its forms shows itself to be quite independent of *climatic influences* in a higher degree even than typhus. The disease has been prevalent with equal frequency and in equal intensity in all latitudes of the Eastern Hemisphere; and if many tropical or sub-tropical districts, especially of the Western Hemisphere, have been hitherto exempt from relapsing fever (or appear to have been), yet, when we consider the general diffusion that the disease has attained in India, the reason of this is to be sought, clearly, not in climatic or such-like circumstances, but more probably in the fact that the indigenous seats of relapsing fever are limited within narrow circles, and that the importation of it from these to other localities is dependent on conditions which do not often occur. In favour of this we may take the fact that even large regions of Europe, standing in the closest and most active intercourse with the districts lately invaded by relapsing fever in Russia, the

[1] Richter, 'Berl. klin. Woch.,' 1877, p. 237.

East of Germany, and Great Britain, and showing no appreciable differences from these in circumstances of climate, have nevertheless remained untroubled by the disease.

§ 146. Little Influenced by Weather.

The absolute independence of the origin of the disease on climate is expressed also in the relation of relapsing fever (as an epidemic) to *influences of season and weather*, which, as Murchison rightly says,[1] comes out much less markedly than in typhus fever, if it exist at all. In 35 epidemics in the United Kingdom, Russia, and Germany, for which we have more particular data as to the time of prevalence, the height of the epidemic was reached twelve times in winter and twelve times in summer, or equally often in the seasons opposite as regards climate; and it was prevalent, further, just as often in warm and moist weather as in cold and dry. If the observations of English practitioners on bilious typhoid on the Mediterranean coasts are in favour of its prevalence in summer, Engel, again, has observed it in Bukowina chiefly in winter (December and January), while Griesinger places the maximum of epidemics in Egypt in spring (February and March). Of six epidemics of relapsing fever and bilious typhoid observed at various parts of India, three came to a height in the hot season (1859 at Saugur, 1865 at Bangalore, and 1877 at Bombay) and an equal number in the cool season (1856-57 at Patna, 1860 at Gazipoor, and 1871 at Kasalong). The following table (p. 608) shows how independent the amount of disease is of seasonal influences.

§ 147. No Relation to the Soil.

It is highly improbable, judging by the available data, that the origin or diffusion of relapsing fever stands in any relation to *the influences of the soil*, to altitude, configuration, the kind of geological formation, and the like. At all

[1] L. c., p. 292.

Table of Cases of Relapsing Fever in the several Months of the Year.

Place.	Years.	Months.												Where treated.
		January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	
St. Petersburg	1865—1869	1462	1390	1345	1230	1105	944	720	547	411	712	874	1152	Obuchoff Hospital.[1]
Leith . .	1843	—	—	—	—	—	—	—	—	144	417	331	224	The Fever Hospitals of the respective towns.
	1844	133	45	—	—	—	—	—	—	—	—	—	—	
Glasgow . .	1843	—	—	—	—	510	1143	1284	3649	2990	1930	1275	1370	
	1847	192	181	265	226	226	229	239	198	182	162	133	107	
Edinburgh .	1843	—	74	83	96	133	161	251	392	531	638	586	544	
	1844	465	300	256	93	50	—	—	—	—	—	—	—	
Riga . .	1864—1875	342			239			563			517			The Town Hospital.[2]

[1] Herrmann, 'Petersb. med. Zeitschr.,' 1870, p. 415.

[2] Girgensohn, l. c., p. 35.

events, in this disease as in typhus, there is not the slightest connexion discoverable between the pathogenesis and *malarious soil*. The fact that relapsing fever is equally prevalent at all seasons, generally during summer and winter, or in India in the hot and cold seasons, and therefore in states of the weather in which malarial influences make themselves felt least, affords an indirect proof against the assumption often made of a connexion of that sort. A direct proof is furnished by the endemicity of the disease in countries, such as Ireland and Scotland, which are distinguished by their immunity from malaria, or cannot, at least, be counted among the proper malarious regions. It is an interesting fact that relapsing fever, imported, as we have seen, into Réunion from India, broke out at a time (1865) when the island enjoyed a complete immunity from malaria, and that it died out[1] the year following, just as an intense focus of malaria developed in the island, as already described (p. 232).

§ 148. Relation to Overcrowding and Privations.

But the most striking analogy to typhus, which relapsing fever manifests, comes out in the relation of the origin of the disease to all those conditions of *social misery* which play so decided a part in the typhus history.

Especial stress has been laid in this connexion on *want of food*, more especially on the ground of observations made in Ireland; and relapsing fever has been called the true "typhus famelicus." It was Murchison[2] who first said "epidemics of recurrent typhus always appear under the influence of want or of hunger;" and he adds[3] that "its origin is much less dependent on overcrowding than spotted typhus, and *that it is more likely to be the result of want alone.*" This view has found confirmation in some of the later epidemics, such as that of 1867-68 in Finland, and those of 1865 and 1877 in certain districts of India, in so far as the outbreak

[1] Azéma, l. c.; Silliau (l. c., p. 21) remarks that the relapses readily gave occasion to a confusion of the disease with malarial fever, and adds: "Si je m' étais trouvé dans un pays paludéen, plusieurs fois je me serais trompé."

[2] 'The Continued Fevers of Great Britain.'

[3] Ib.

of the disease has in fact coincided with starvation among the people; but by far the larger number of observations do not show any such direct connexion between the sickness and this etiological factor.

At the first breaking out of the disease in Russia, the St. Petersburg physicians[1] stated that there could be no idea of famine or even of scarcity as the cause of the disease, and the later observations in that country point to the same conclusion. "The relapsing fever," says Herrmann,[2] "was by no means brought on by actual famine in St. Petersburg; such a thing is quite unknown with us. . . . In the west, also, whither the disease travelled from Russia, it reached its height, not in famine years, but in good times; as with us, it was the working classes that suffered almost exclusively, and our patients were seldom starving but usually well-nourished, healthy people." To the same effect is the *resumé* of Girgensohn[3] from his experiences made in the Riga epidemics: "I do not consider myself justified in assigning to circumstances of nutrition a material part as etiological factors in our epidemic, and the less so that not only did the majority of our patients appear to be in a tolerably good state of nutrition, but also that the observations in the prison went entirely against such an assumption." In England and Scotland, also, the conviction has subsequently gained ground that Murchison's theory is untenable. Thus, Muirhead says of the Edinburgh epidemic of 1870:[4] "I wish to state that in no single instance which came under my observation could starvation be said to be the immediate cause of the disease. Not one of those individuals could be said to be emaciated. In fact, they were all wonderfully clothed with fat. . . . On strict and repeated inquiry, not one of them would confess to having been in destitute circumstances." Neither could there be any question of scarcity or famine in other parts of Scotland in 1870; and the same applies to England. Thus, Rabagliati says of the epidemic at Bradford and Liverpool: "It must be said that careful inquiry in Bradford often failed to elicit the fact that the sick had had to undergo any special hardships, or to endure any special privations in food; trade was moderately good in the town and the consequent demand for labour pretty active. In Liverpool, at the time of the epidemic there was full employment for every able-bodied and industrious man. . . . From all these circumstances it appears that the name of famine fever is not in all circumstances a quite appropriate one." To the same effect is the expressed opinion of those who saw the epidemics of 1868 and 1872 in Breslau[5] and Berlin (Obermeier, Budberg, and others), as well as of the writers on relapsing

[1] Zorn and others.

[2] 'Petersb. med. Zeitschr.,' 1870, p. 421.

[3] L. c., p. 37.

[4] L. c., p. 5.

[5] See particularly Litten, l. c., p. 139, and the explanation by Wyss and Bock referred to in the sequel.

fever in the United States; thus Parry says, with reference to the epidemic of 1869-70 in Philadelphia, that there was no want of the means of subsistence at the time, and that all the patients received into the hospital with relapsing fever were well nourished.

It cannot be doubted that the weakening of the organism, or the lowering of its power to resist injuries from without, is an essential predisposing cause of sickness from relapsing fever as well as from typhus. In this sense we have to read the experiences of Ireland, of India, and other countries, and it has been pointed out also by Wyss and Bock for the 1868 epidemic in Breslau.

"The outbreak and spread of the epidemic coincided," they say,[1] "with the time when the means of subsistence had reached their highest price and their lowest consumption; or, to speak more correctly, the disease breaks out and spreads among a population reduced and weakened by deficient nourishment, and less able than in 'good times' to resist external influences, particularly contagion. We cannot venture to conclude that the relapsing fever was the direct result of want, that the want of food engendered the morbid contagion, or that, in consequence of want, a febrile condition was produced in many persons, out of which relapsing fever developed. We lay stress on the fact that the scarcity in the particular quarters of the town reached by no means so high a point that men sickened solely in consequence of hunger."

This etiological factor, then, certainly plays no specific part in the genesis of relapsing fever. Rather, the proper soil is formed for the development and spread of the disease by all those *defects in the conditions of residence and livelihood*, which give occasion to the development and accumulation of putrid decomposition-products, and, above all, by crowding into filthy and ill-ventilated rooms which, as we have seen, are the specific breeding-places of the typhus poison also. It is this that explains the *association of relapsing fever, as well as typhus, almost exclusively with the proletariat.* In the epidemiographical accounts before us, from Europe and the United States, there is almost complete agreement that the disease has attained its epidemic diffusion only among those sections of the community that are particularly ill-situated in matters hygienic, in the over-populated and filthy quarters or houses; that in the large towns of England, Scotland, and

[1] L. c., p. 26.

Germany (for instance, Berlin), the lodging-houses and taverns of the lowest grade have been the headquarters of the disease and very often the points of departure of an epidemic; that, where sporadic cases have occurred, as in many parts of Germany (Greifswald, Swinemünde, Danzig, Magdeburg, Leipzig, Dresden, Brunswick, and Giessen), it has been chiefly if not exclusively travelling artizans and vagabonds who have made up the list of the sick; that among the classes of the populace in more comfortable circumstances, cases of sickness from relapsing fever have been extremely rare, and in many epidemics have not been observed at all; that relapsing fever, therefore, as Engel says of it in the Bukowina, is peculiarly a "morbus pauperum." All experience as to the occurrence and diffusion of the disease on African and Asiatic soil has led to the same conclusion.

In Egypt, Griesinger[1] has seen the disease only among the lowest orders, among soldiers, labourers, and the like; among the classes in better circumstances, and particularly among the Europeans living in the country, no case of bilious typhoid came under his notice. In Réunion, according to the confirmatory accounts of Silliau[2] and Coustan,[3] it was only the negroes, coolies, and natives living in crowded and filthy huts that suffered; the whites, living in comfortable circumstances, were quite exempt. In India, also, the sickness has confined itself to the proletariat; the *prisons* have been its head-quarters, whence it has spread subsequently to the population at large. The same fact —outbreak of the disease as a prison fever—has been observed also at other places, as at Tarnopol[4] in 1865, at Constantine[5] in 1867, and at Hong Kong[6] in 1865; so that, in this respect also, there is a striking analogy, in the morbid development, between typhus and relapsing fever (including bilious typhoid).

§ 149. Question of its Spontaneous Origin or of its Parasitic Nature.

Just as recent experience has shown that Murchison's view of the direct production of relapsing fever in the individual by want of food, is untenable, so there is equally little reason to seek for the proper source of the morbid poison in the unhygienic conditions of which we have been

[1] L. c., p. 56. [2] L. c., p. 18. [3] L. c., p. 21.
[4] Leiblinger, l. c. [5] Arnould. [6] Murray.

speaking, or to see in them more than a peculiarly favorable soil for the poison to grow in. Social misery, and its consequences, have always existed, and at all parts of the world; but the area of distribution of relapsing fever and bilious typhoid, although it may be more extensive than we have hitherto known it, is still a very limited one. But the question of the (so-called) *spontaneous origin* of the disease, or of its virus, out of any or all of the injurious influences before mentioned, can hardly be discussed seriously at the present day. Not only has the constant occurrence of an organism of the lowest grade of development—the spirillum or spirochæta—been made out in the blood of patients with relapsing fever or bilious typhoid, but also the specific relation of it, as the proper cause of the disease, has been established with at least a high degree of probability. We have thus obtained, not only an exact proof of the *specificity and the parasitic character of this disease* and of the *identity of relapsing fever and bilious typhoid*, but, more generally, we have found a basis, in a measure secure, for the theory of the *parasitic nature of the so-called infective diseases.*

Obermeier, as is well known, has the merit of having first made out the spirillum in the blood of relapsing-fever patients, and of having correctly estimated its relation to the morbid process. His pioneering work[1] was followed by other researches on the parasite, confirming and extending it, on the part of Engel,[2] Weigert,[3] Birch-Hirschfeld,[4] Heidenreich,[5] Moschutkowsky,[6] Carter,[7] Albrecht,[8] and Koch.[9] Moschutkowsky was the first to attempt to produce infection by inoculating the blood of relapsing-fever patients on healthy men, and he proved not only the communicability of the disease, but also the specific pathogenetic significance of the parasite. Inoculations on lower animals

[1] 'Centralbl. für die med. Wiss.,' 1873, No. 10; and 'Berl. klin. Woch.,' 1873, Nos. 33, 38.

[2] Ib., 1873, No. 35.

[3] Ib., 1873, No. 49, 1874, No. 5; 'Deutsche med. Woch.,' 1876, Nos. 40—42.

[4] 'Arch. für klin. Med.,' 1874, xiii, p. 346.

[5] 'Petersb. med. Woch.,' 1876, No. 1; 'Klinische und mikroskop. Untersuchungen über die Parasiten des Rückfalltyphus,' Berlin, 1877.

[6] 'Centralbl. für die med. Wissensch.,' 1876, No. 11; 'Petersb. med. Woch.,' 1878, No. 27; 'Arch. für klin. Med.,' 1879, xxiv, p. 80.

[7] 'Brit. Med. Journ.,' 1877, Nov., p. 700; and 'Med.-Chir. Transact.,' 1878, lxi, p. 273.

[8] 'Petersb. med. Wochenschr.,' 1878, No. 20.
According to Cohn, 'Deutsche med. Wochenschr.,' 1879, Nos. 16, 27, 30.

(dogs, rabbits, and the like) failed with him, as with Carter; but the latter, as well as Koch, experimented successfully on apes, in so far that the infected animals were attacked with intense fever a few days after the inoculation had been made, and spirilla were found in quantities in the blood. Finally, there is an especial interest in the fact made out by Moschutkowsky, that the parasite of relapsing fever occurs likewise in the blood of individuals suffering from bilious typhoid, and that experiments to infect healthy men with the blood of patients suffering from that form of the sickness, have resulted in an attack of relapsing fever; so that the identity of the two diseases, already maintained by Griesinger and the Russian physicians in respect of the anatomy and pathology, may be inferred with a high degree of certainty.

§ 150. A Communicable Disease.

As to the *native habitat of relapsing fever*, involving the question whether it is as wide as the distribution-area of the disease itself, or whether the disease is indigenous at certain points of the globe only, occurring at others merely in consequence of an importation of the morbid poison, and the question where these indigenous foci are to be sought for—on all these questions we are unable for the present to arrive at more than an incomplete judgment, and this is owing more especially to the fact that so little is known of the history of relapsing fever in past centuries. There can be no doubt that the disease is endemic in Ireland; but the opinions of practitioners in Britain are divided as to whether that applies also to England, and more especially to Scotland. Other native foci may be conjectured, with some probability, to exist in Russia (whence the disease has penetrated to Germany and Austria), and in Egypt and India; but we are unable to decide for the present whether there may not be some central focus, in Asiatic territory, between Russia and India.

There exists no doubt whatever in the experience of *all* observers, and according to the experimental inoculations, that relapsing fever (or bilious typhoid) *is contagious*, or, in other words, that the disease is communicable by a morbid poison reproduced in the sick person and eliminated by him in a condition of potency. The most obvious proof of its conveyance from place to place and from individual to individual

has been furnished by the breaking out of the disease at isolated spots on the arrival there of the sick,[1] as well as by the spreading of the disease to the attendants and other persons in hospitals.[2] In favour of this, also, is a series of observations to the effect that not merely the patient himself, or the atmosphere surrounding him forming the medium of the poison, but even healthy persons living in the vicinity of relapsing-fever patients, or the articles which the patients may have used, may become carriers of the morbid poison.

"From the number of laundry-women that have been attacked," says Cormack of the epidemic of 1842 in Edinburgh,[3] "it appears that the clothes of our fever patients are especial repositories and communicators of the morbid poison." Donbowitzky also, for St. Petersburg, mentions the conveyance of the disease by infected articles of clothing. Messrs. Wyss and Bock adduce, from the Breslau epidemic of 1868, a series of observations [4] which seem to prove the protracted clinging of the morbid poison to rooms that had been occupied by the sick, as well as the conveyance of the disease by healthy persons and by effects (here also especially in washerwomen). Further, there are two really classical examples given by Parry[5] from the epidemic of 1869 in Philadelphia, of infection by articles of clothing which had been worn by relapsing-fever patients during their illness. Clark relates that the first case of relapsing fever in New York in 1870 occurred in a house which was the seat of a brisk old-clothes trade between that city and London, the conjecture naturally arising that the morbid poison had been imported with these articles from London, where the disease was then epidemic.

§ 151. No Preference for Race or Nationality.

Finally, it should be mentioned that circumstances of *race and nationality*, have been entirely without influence on the

[1] There is an interesting example of this in the epidemic outbreak of relapsing fever in Réunion, on the arrival of an English vessel from Bombay with coolies, who had contracted the disease in that city. The fever was afterwards carried from the shore to two vessels at anchor in the port of St. Denis (Normand, 'Hygiène et pathologie de deux convois de condamnés aux travaux forcés transportés de France en Nouvelle Caledonie,' Paris, 1869).

[2] There are observations of this kind from hospitals in St. Petersburg (Herrmann and Küttner), Breslau (Wyss and Bock), Edinburgh (Craigie), Danzig (Knipping), New York (Harris), Patna (Sutherland), Chittagong (Lyons), the Punjaub (Murray), and Réunion (Silliau, Azéma).

[3] L. c., p. 117. [4] L. c., p. 51 ff. [5] L. c., p. 341.

diffusion of the disease, wherever it has appeared as an epidemic. As the experiences of Europe teach us, the exemption which the European residents have enjoyed in Egypt, India, Hong Kong, Réunion, and other extra-European countries is not referable to peculiarities of race, but solely to the favorable circumstances in which they live as compared with the negroes and poor natives of those regions.

CHAPTER XIII.

TYPHOID FEVER.

(Typhus abdominalis, or Ileo-typhus.)

§ 152. Confused Identity in Earlier Writings.

The doctrine of the so-called "abdominal typhus" counts among the acquisitions which medicine owes chiefly to the methodical investigation of morbid anatomy. Its precise establishment is a product of modern times; it saw the light after severe labour, as we may learn from the keen controversies over the term "typhus" among French, English, and German physicians, in the fourth and fifth decades of this century. Historical inquiry, accordingly, into the relations of typhoid in time and place can only cover a somewhat short period with any exactness, a period counted by tens of years. But in the history of pestilence in past centuries there are still a good many indications, more or less ambiguous, of the general diffusion of the disease in former times as well; and these, taken along with the experiences of the present, justify the conclusion that typhoid takes a leading place among those acute infections which bear the pronounced character of ubiquitous diseases.

In the medical writings of antiquity and the middle ages we seek in vain for descriptions or even moderately trustworthy indications of the group of symptoms peculiar to typhoid. It stands to reason that we may not conclude therefrom that the disease occurred rarely in those times, or not at all;[1] on the contrary, there are, in several writings of the sixteenth and seventeenth centuries on the "febres pestilentes"

[1] See the thorough handling of the history of typhus abdominalis down to the beginning of the nineteenth century, in Häser's 'Lehrbuch der Geschichte der Medicin,' 3rd ed., vol. iii, p. 575 *et seq.*

then observed, accounts of certain forms of sickness which can hardly be interpreted otherwise than as referring to typhoid; and still more frequently and more plainly do we encounter the lineaments of this disease in the epidemiographical and clinical literature of the eighteenth century, in which there are not wanting even some anatomical notes relating undoubtedly to typhoid.

Not to mention the somewhat ambiguous statements of Spigelius, Panarolus, and other observers of the sixteenth and seventeenth centuries, in which Murchison[1] thinks that he can detect the description of typhoid, the first tolerably definite indications of the disease that we meet with occur in the description given by Willis of "febris putrida maligna" in his treatise on fevers;[2] further, in Sydenham's sketch of the "peculiar" kind of malignant fever observed by him in London in 1661-64;[3] again, in the account by Baglivi[4] of the form of fever which he described under the name of "febres malignæ et mesentericæ;" in Lancisi's[5] account of the condition found on dissection in the "febres perniciosæ" that were prevalent in Rome, 1695; and in Hoffmann's[6] notes of his epidemiological observations on "febres epidemicæ, exanthematicæ catarrhales, sive petechizantes" at Halle in 1698—1728.

Among the writings of the eighteenth century, especial mention has to be made of Strother's[7] account of "slow" or "lent fevers" which were prevalent in London in 1727-29, their proper (anatomical) cause, according to him, being inflammation and ulceration of the intestine; further, of Gilchrist's[8] description of the "nervous fever" which he saw at Dumfries in 1735; the work by Chirac[9] on

[1] 'The Continued Fevers of Great Britain,' Lond., 1862.

[2] 'Lib. de febribus,' cap. x, xiv, Opp. Amstelod., 1682, pp. 82, 110.

[3] 'Observ. med.,' i, cap. 4. Greenhill's ed. (Syd. Soc.), p. 39.

Prax. med.,' lib. i, Opp. Antwerp, 1715, p. 51.

'De noxiis paludum effluviis,' lib. ii, epid. i, cap. vi, Colon. Alloborg, 1718, p. 160.

[6] 'Medicina rationalis system.,' tom. iv, sect. i, cap. 10, Opp. Genev., 1748, ii, p. 75.

[7] 'Pract. Observ. on the Epidemical Fever,' Lond., 1729, pp. 15, 164.

[8] 'Med. Essays and Observ., published by a Society in Edinburgh,' Germ. transl., iv, p. 453, v, p. 654.

[9] 'Traité des fièvr. malignes,' Paris, 1742, p. 50.

malignant fevers, in which he calls attention to the morbid changes appearing in the intestinal mucous membrane in the course of them; and the masterly account of "slow nervous fever" by Huxham.[1] To this period, also, belongs the description by Morgagni[2] of the *post-mortem* condition in cases of bloody intestinal discharges, most of which were dysentery, while some of them were clearly typhoid. In German medical literature the first undoubted description of typhoid occurs in the small work of Riedel[3] on "intestinal fever," following which we have the classical work of Röderer and Wagler[4] on the epidemic of "mucous fever" at Göttingen in 1760,[5] and the work of Riepenhausen treating of the same epidemic. Further information on the occurrence of typhoid, along with typhus and dysentery, in Italy, may be found in the accounts by Sarcone,[6] Pepe,[7] Fasano,[8] and others on the severe famine-fever which was prevalent in Naples in 1764.[9] The disease appears to have played no inconsiderable part, also, according to indications by Mayer,[10] Wienholt,[11] and others, in the famine-fevers of Germany in 1770-72. The same is true of the mucous fevers, bilious fevers, and putrid fevers in Germany, of which there are many epidemiographical accounts[12] dating

[1] Opp. Lips., 1784, i, p. 163, ii, p. 78.

[2] 'De sedibus et causis morbor.,' lib. iii, Epist. xxxi, Venet., 1761, ii, p. 25.

[3] 'Progr. de febr. intestinal.,' Erford., 1748. Reprinted in Baldinger, 'Sylloge,' i, p. 42.

[4] 'De morbo mucoso liber singularis,' Gott., 1783.

[5] 'Morb. epidem. . . . 1757–62 Gottingæ et circa eam grassati.,' Hal., 1766.

[6] 'Istoria ragionata dei mali osservati in Napoli nell' intero corso dell' anno 1764,' Napoli, 1765.

[7] 'Il medico di letto,' Napoli, 1766.

[8] 'Della febre epidemica sofferta in Napoli l'anno 1764,' libri iii, Nap., 1765.

[9] A detailed account of this destructive pestilence has lately been published by De Renzi ('Napoli nell' anno 1764, ossia documenti della carestia e della epidemia che desolarono Napoli nel 1764,' Napoli, 1868). See also Corradi, 'Annali delle epid. occorse in Italia,' iv, p. 205, *seq.*

[10] 'Abriss der Epidemie zu Jena,' Jena, 1772.

[11] 'Diss. de inflammationibus viscerum hypochondriac. occultis in febribus biliosis putridis,' Gotting., 1772.

[12] See Finke on the epidemic of 1776 in the County of Tecklenburg ('De morbis biliosis anomalis,' Monaster.,' 1780, p. 21); Cless, on the Stuttgart epidemics of 1783 and 1792 ('Geschichte der Schleim-fieber-Epidemieen Stuttgarts,' Stuttgart, 1837, p. 2, after the college essays of Consbruch and Kraus, and of Jacobi); and Eckner, on an epidemic in 1789 in the neighbourhood of Rudolstadt ('Beitrag zur Gesch. epid. Gallenfieber,' Leipzig, 1790).

from the last twenty years of the eighteenth century; and it is especially true of the "war fevers" which were prevalent in Germany and France at the end of the last century and beginning of the present.[1]

From Great Britain there are not less numerous notices of typhoid in those years; for example, by Sutton[2] on an epidemic at Deal in 1806, by Bateman[3] on the prevalence of the disease in London, by Muir[4] on an epidemic in a suburb of Paisley in 1811, and by Edmonstone[5] on the disease in Newcastle in 1817.

All these and many other notices of that period had so far advanced knowledge as to furnish incontrovertible proofs of the intimate connexion between certain forms of febrile sickness and severe affection of the intestinal mucous membrane. But it was not until an impulse was given to pathology by the morbid anatomy of the Paris school, that a clear recognition of the *peculiarity of the form of fever* was arrived at. Prost[6] had already declared an intestinal affection to be a constant symptom of "fièvres ataxiques;" but his notion of "fièvres ataxiques" was a very vague one, and in the clinical histories, as well as in the *post-mortem* records, which he has collected under that title, there is a good deal of heterogeneous matter thrown together. Petit and Serres were the first to recognise accurately the charac-

[1] Canz, on the epidemic of 1793 in Hornberg, Black Forest ('Beschreibung einer Schleim-, Faul-, und Nervenfieber-Epidemie,' Tübingen, 1795; Eisfeld, on the epidemic of 1799 at Leipzig ('Meletemata ad historiam typhi acuti Lipsiæ 1799 grassati,' Lips., 1800, 1801); Reinhard, on the Wittemberg epidemic of 1807-8 ('Diss. de febre nervosa,' Viteberg., 1809); Wittmann, on the epidemic of 1806 and following years in and around Mainz ('Die neuesten am Rhein herrschenden Volkskrankheiten,' Mainz, 1811, p. 27); Weber, on the epidemic of 1814 in Kiel and vicinity ('Bemerk. über die in Kiel . . . 1814 vorherrschenden Krankheiten,' Kiel, 1814); Heusinger, on the sickness among the German troops quartered in 1817 at Thionville, Metz, and other places in Lorraine ('Rust's Magaz. für Hlkd.,' 1819, v, p. 201).

[2] 'Account of a Remittent Fever among the Troops in this Climate,' Canterbury, 1806.

[3] 'Account of the Contagious Fever of this Country,' Lond., 1818, p. 145.

[4] 'Edin. Med. and Surg. Journ.,' 1812, April, p. 134.

[5] Ib., 1818, Jan., p. 174.

[6] 'Médecine éclairée par l'observation et l'ouverture des corps,' Paris, 1804, i, p. lvi: "I have opened more than two hundred bodies of persons who have died in the course of ataxic fevers, and I have always observed inflammation of this membrane (the intestinal mucous membrane)."

teristics of typhoid and to state precisely the symptomatological and anatomical peculiarities of the disease. Their treatise,[1] in which they record the results of their observations gathered from numerous patients in the Hôtel Dieu, was followed by the works of Cloquet, Andral, Bretonneau,[2] and lastly the classical memoir of Louis,[3] who introduced the designation of "fièvre typhoide" which has been in general use in France since that time and was subsequently adopted in England.

Meanwhile, Pommer[4] had been attempting by his small work on 'Sporadic Typhus,' to attract the attention of German physicians to the disease in question, and particularly to determine its anatomical characters from his own observations. His book, however, like those that followed it on the same subject, by Heusinger, Lesser, and others, did not receive so much notice as it deserved; and it was not until the advent of Schönlein, who introduced the name of "abdominal or ganglionic typhus," and until the writings of the Vienna school, that correct opinions upon typhoid became somewhat general in Germany; so that finally an understanding was arrived at on the specific differences between this disease and exanthematic typhus—a disease which had almost passed out of memory there, as it had done also in France since the Napoleonic wars. The delay in clearing up the confusion about typhoid was longest in Great Britain; in that country also, between 1820 and 1850, there was no lack of observations on the disease or of descriptions of it, but only a few observers had correctly recognised its peculiarity; it was for the most part included with typhus under the comprehensive notion of "continued fever," and Jenner, in his classical work,[5] was the first to resolve this notion into

[1] 'Traité de la fièvre entéro-mésentérique,' Paris, 1814.

[2] The first account of the disease which he observed at Tours in 1826, and named "dothiénentérite," was given by Trousseau in the 'Arch. gén. de méd.,' 1826, x, pp. 67, 169.

[3] 'Recherches anatomiques pathol. et thérap. sur la maladie connue sous les noms de gastroentérite, fièvre putride, &c.,' Paris, 1829.

[4] 'Beiträge zur näheren Kenntniss des sporadischen Typhus,' Tübingen, 1821.

[5] The papers appeared first in the 'Medical Times,' 1849–51; afterwards in a French translation by Verhaeghe ('De la non-identité du typhus et de la fièvre typhoïde,' Bruxelles, 1852, 1854).

its elements and to introduce into England a general *éclaircissement* of the so-called "typhoid diseases."

It was inevitable that, with this light thrown upon the nature of a disease that was so common and so serious, the medical world should apply itself to the study of typhoid with a peculiar interest; and so there quickly grew up, first in France, later in Germany, England, and all other civilised countries, an epidemiographical literature of typhoid in which the individual papers previous to 1860 were counted by thousands, and which has experienced a scarcely less remarkable growth in the twenty years following. This large amount of writing on typhoid, especially between 1830 and 1840, has given rise to the often expressed opinion, which I shared at one time, that we have to do with an unusually general prevalence of the disease during that period, or that its general diffusion had not been reached until recent times. I think, however, that we must consider this to be an erroneous view, and that we must explain the phenomenal outburst of literature by the sudden and rapidly culminating interest of the profession in a new and important object of study—a phenomenon which has recurred in the case of many other forms of disease. Without denying that changes in hygienic conditions have not been without influence on the increase or decrease in the amount of typhoid at many parts of the globe, we may virtually ascribe the appearance of a general increase in the amount and diffusion of the disease in recent times to the circumstance that the literary material which an inquiry into typhoid can collect from past centuries is somewhat scanty. This want of epidemiographical notices about typhoid, should be considered to be only apparent, as we have already shown; it is extremely probable that, behind all those febres pestilentes, malignæ, putridæ, nervosæ,—behind the mucous and bilious and putrid fevers of former times—the disease in question, typhoid fever, lies concealed; and this conjecture, as we shall see, finds support in the history of typhoid in India.

[1] 'Prager Viertelj. für die pract. Hlkde,' 1852, ii, p. 37, and in the 1st edition of this work, vol. i, p. 158.

§ 153. Present Geographical Distribution.

On the soil of *Europe* typhoid takes one of the foremost places among the acute infective diseases, in respect of the frequency of its occurrence and the range of its diffusion. In *Iceland* the disease, known by the name of "Landfarsot" (sickness of the country), is met with almost every year, and not unfrequently in epidemic form and of a malignant type.[1] It has the same character in the *Faröe*[2] and *Shetland Isles*;[3] and the authorities give the same accounts of its endemic and epidemic prevalence in *Norway*,[4] *Denmark*,[5] *Sweden*,[6] *Finland*,[7] and *Russia*.[8]

[1] See Manicus, 'Bibl. for Laeger,' 1828, i, p. 207; Thorstensen, ib., 1838, i, p. 293; Schleisner, 'Island undersögt fra et laegevidenkabeligt synspunkt,' Kjöbenh., 1849, p. 46; Hjaltelin, 'Edin. Med. Journ.,' 1872, Feb., p. 710; Finsen, 'Jagttagelser angaaende sygdomsforholdene in Island,' Kjöbenh., 1874, p. 15.

[2] Panum, 'Bibl. for Laeger,' 1847, Jan., p. 313. See also the accounts of its epidemic outbreaks in 'Sundhedskollegii Aarsberetninger,' 1853, p. 29, 1857, p. 294, 1858, p. 421.

[3] Sexby, in 'Dobell's Reports,' 1870, ii, p. 522.

[4] Kjerulf, 'Verhandl. der Würzb. phys.-med. Gesellsch.,' 1852, iii, p. 37; Eger, 'Norsk Magaz.,' 1870, p. 406; Broch, 'Le Royaume de Norvège, &c.,' Christiania, 1876, p. 55; Larsen, 'Om forekomst af tyfoid feber i Norge indtil 1876,' Christiania, 1879 (Supplement to the 'Norsk Magaz. for Laegevidensk, 1879), according to statistical reports for the years 1857–75. See also the epidemiological account by Sandberg, 'Norsk Magaz. for Laegevidensk,' viii, p. 257 (for the year 1837 in Friderikshavn); of Corradi, ib., vii, p. 227, 1860, p. 14, 1861, p. 501, 1862, p. 714, 1864, p. 902 (epidemics in Christiania); of Homann and Hartwig, ib., 1865, p. 433, and Daae, ib., 1868, p. 13 (epidemic of 1864 at Krageröe); of Collet, ib., 1868, p. 609 (epidemics from 1864 to 1867 in the district of Nordland); and of Holst, ib., 1869, p. 13 (epidemic of 1866–68 at Drammen).

[5] See the official reports published annually in the 'Sundhedskollegii Aarsberetninger,' from 1846 onwards.

[6] Huss, 'Om Sverges endem. sjukdomar,' Stockh., 1852, *passim*, and his 'Statistique et traitement du typhus et de la fièvre typhoide,' Paris, 1855; Berg., 'Bidrag til Sveriges med. topogr. och statistik,' Stockh., 1853, pp. 23, 201. See also the epidemiographical accounts in 'Svenska Läk. Sällsk. Handl.,' and the excellent sanitary reports ('Sundhets-Kollegii Berättelse') which have appeared annually since 1852.

[7] Ilmoni, Ringbom, and others in 'Finska Läk. Sällsk. Handl.,' i, *et seq.*

[8] From all parts of this vast aggregate of countries there are accounts of "typhus epidemics," or of the endemic occurrence of "typhus;" but in many of them it is doubtful whether typhus exanthematicus or typhoid is meant. Relating certainly to the latter, there are many accounts: from the Baltic Provinces (Sahmen, 'Petersb. med. Abhandl.,' iv, p. 38; Gramkau, 'Hamb. Zeitschr.

Broch estimates that an average of three per thousand of the inhabitants of *Norway* suffer annually from typhoid; Eger and Larsen agree in saying that the disease is more frequent and more widely spread in the western or coast regions than in the eastern or inland, the annual number of cases per thousand of the inhabitants being 5·1 in Tromsöe, 3·3 in Trondjhem, 3·3 in Bergen, 3·0 in Christiania, 2·7 in Christiansand, and 1·5 in Hamar. In *Denmark*, according to official reports over fifteen years (1846-60), there have been more than one hundred larger or smaller epidemics of typhoid at various parts of the country. In *Sweden*, according to official reports for the period from 1855 to 1877, there have been some 200,000 cases of typhoid, or more than 10,000 annually, which have come to the official cognisance; or, estimating the mean population of the country during the nineteen years at 4,000,000, the rate of sickness has been nearly three per thousand.

In *Germany*, the *Netherlands*, *Belgium*, *France*, and *Switzerland*, typhoid, as is well known, counts among the standing diseases; and it is not less general as an endemic and epidemic in *Great Britain*, particularly in England and Ireland, and in the West of Scotland, other districts of Scotland appearing to be somewhat exempt.[1]

For *Belgium* Ridder[2] estimates the annual deaths from typhoid, on an average of the years from 1851 to 1870, at 4·1 per cent. of the total mortality. From *Switzerland* we have numerous epidemiographical accounts of typhoid in all the cantons, as well as information as to its endemic prevalence in several districts of the Canton Zürich,[3] in Basel,[4] in Geneva,[5] and other places.[6] In France, according to the statistical *resumé* furnished to the Academie de Médecine by Magne,[7] there were 757 epidemics of typhoid in the period from 1841 to 1863 (excluding 1858). The departments most affected come in the following order: Jura (36 times), Haut-Saône (29), Doubs and Moselle (each 28), Nord and Pas-de-Calais (each 26), Aisne (23), Côte-d'Or (22), Seine-et-Marne (21), and Saône-et-Loire (20). The least affected departments were: Calvados, Eure, Haute-Garonne, Finistère, Landes, Loire, Loiret, Maine-et-Loire,

f. med.,' vi, p. 419), from St. Petersburg (Thielmann, 'Med. Jahresbericht,' 1840, p. 3, 1841, p. 23, and 'Med. Ztg. Russl.,' 1846, No. 9, 1847, No. 40), from Tula (Koch, ib., 1857, p. 9), from Podolia (Passower, ib., 1845, No. 9), from the Caucasus (Liebau, 'Petersb. med. Zeitschr.,' xi), and from Kutais (Krebel, 'Med. Ztg. Russl.,' 1858, p. 76).

[1] Murchison.

[2] 'Annal. de la Soc. de méd. de Gand.,' 1880, Févr., p. 37.

[3] Wegelin, 'Der Typhus im Kanton Zürich,' Zürich, 1854.

[4] Socin, 'Typhus, Regenmenge und Grundwasser in Basel,' Basel, 1871.

[5] Marc d'Espine, 'Schweizer Zeitschr. für Med.,' 1849, p. 1.

[6] See Lombard, 'Climatologie méd.,' iii, p. 24.

[7] 'Bull. de l'Acad. de méd.,' 1865-66, xxxi, p. 94.

Puy-de Dôme, and Bas-Rhin (each 4 times); Ain, Arriège, Avignon, Creuse, Manche, Haut-Rhin, and Yonne (each 3 times); Ardennes, Bouches-du-Rhône, Aude, Cher, Eure-Loir, Rhône, Var, and Haute-Vienne (each twice); and Indre, Basses-Pyrénées, and Vendée (each once). The departments of Cantal, Corrèze, and Tarn, had been quite free from typhoid epidemics during those twenty-three years. In the French army the mortality from typhoid in the period from 1862 to 1875 reached a mean annual average of 2·23 per thousand troops.[1]

As to the relative frequency of the disease in the several counties of *England* we have the following details from the official statistics.[2] From 1869 to 1877 there were registered in *England and Wales* (with a population of 23,000,000 in round numbers, according to the census of 1871), 73,859 deaths from typhoid, the mean annual mortality from the disease over the whole country being, therefore, 3·7 per 10,000 inhabitants. The following table shows the relative yearly mortality in the several counties and in the capital.

Table of the Mean Annual Mortality from Typhoid in English Counties and the Metropolis, 1869-77.

County or District.	Mean population.	Number of deaths from typhoid.	In 10,000 inhabitants.
1. Suffolk . . .	348,000	710	2·2
2. Sussex . . .	420,000	871	2·3
3. Buckingham . .	160,000	366	2·5
4. Dorset . . .	190,000	411	2·5
5. Cambridge . .	192,000	451	2·6
6. Shropshire . .	267,000	618	2·6
7. Surrey . . .	365,000	872	2·7
8. Bedford . . .	152,000	362	2·7
9. Norfolk . . .	432,000	1053	2·7
10. Wiltshire . .	245,000	585	2·7
11. Essex . . .	442,000	1076	2·8
12. Worcester . .	336,000	855	2·8
13. North Wales . .	441,000	1075	2·8
14. London . . .	3,300,000	7997	2·9
15. Berkshire . .	230,000	595	2·9
16. Middlesex . .	270,000	699	2·9
17. Hertford . .	200,000	524	2·9
18. Somerset . .	484,000	1266	2·9
19. Kent . . .	630,000	1698	3·0
20. Oxford . . .	180,000	425	3·0
21. Gloucester . .	488,000	1298	3·0
22. Hereford . .	110,000	293	3.0
23. Lincoln . . .	428,000	1207	3·1

[1] Colin, 'De la fièvre typhoïde dans l'armée, Paris, 1878, p. 3.

[2] From the Annual Reports of the Registrar-General; typhus and typhoid do not appear as separate causes of mortality until 1869.

County or District.	Mean population.	Number of deaths from typhoid.	In 10,000 inhabitants.
24. Westmoreland . .	650,000	180	3·1
25. Hampshire . .	530,000	1577	3·2
26. Cheshire . .	534,000	1556	3·2
27. Huntingdon . .	59,000	179	3·3
28. Cornwall . .	358,000	1151	3·4
29. Devon . . .	606,000	1913	3·6
30. Cumberland . .	220,000	714	3·6
31. Warwick . .	630,000	2119	3·7
32. Rutland . . .	24,000	79	3·7
33. Northampton . .	250,000	847	3·8
34. Northumberland .	387,000	1390	4·0
35. Derbyshire . .	325,000	1220	4·1
36. Monmouth . .	220,000	820	4·2
37. Stafford . .	877,000	3409	4·3
38. Leicester . .	275,000	955	4·3
39. York, E. Riding .	306,000	1179	4·3
40. South Wales . .	778,000	3099	4·3
41. Lancashire . .	2,850,000	11,366	4·4
42. Nottingham . .	353,000	1704	5·3
43. York, N. Riding .	235,000	1132	5·3
44. York, W. Riding .	1,900,000	9250	5·4
45. Durham . .	742,000	4326	6·5

In the countries of Southern Europe, also, typhoid takes a leading place in the statistics of sickness. Thus in *Italy*,[1] while the deaths from typhoid in the army in 1877 were 342, in a general mortality of 2072 among an effective force of 193,650 men, or 1·8 per 1000, the deaths from this disease in the country generally reached the enormous figure of 16·5 per cent. of the total mortality, or more than half as much again as consumption. In the *Balearic Islands*, according to Weyler,[2] typhoid counts among the most common diseases. In *Malta*, where typhoid is endemic, it breaks out not unfrequently into malignant epidemics.[3] Of the (quondam) *Danubian Principalities*, Lecomte[4] says that typhoid is more or less common and widely diffused every year throughout the whole region, and not unfrequently

[1] An exhaustive collection of the extremely numerous accounts of typhoid epidemics in Italy and Sicily since the beginning of the century will be found in Corradi, 'Annali delle epidemie occorse in Italia,' Disp., iv, p. 453, *et seq.*

[2] 'Topogr. méd. des îles Baléares,' Palma, 1854.

[3] Marston, in 'Statist. Reports of the Army for the Year 1861,' iii, p. 486.

[4] 'Considerations sur la pathologie des provinces du Bas-Danube,' Montpell., 1869, p. 33.

epidemic. From *Turkey* there are accounts of typhoid epidemics by Rigler,[1] Marroin,[2] Mordtmann,[3] and others.

Typhoid in India.—The investigation of typhoid in *Asiatic countries* has had to encounter considerable difficulties owing to the fact that the observers and authorities have only lately arrived at a correct diagnosis of the forms of fever that come under their observation; even the latest information from these regions on the sickness in question suffers from obscurity and palpable error. The history of typhoid in India, which is fully set forth in the sequel, furnishes a striking proof of this, and even justifies the assumption that the disease is much more frequent and widely spread in many parts of Asia than the available data allege, or enable us to infer. Thus the statements of Robertson[4] and Yates,[5] as to the rare occurrence of typhoid in Syria, are balanced by the account of Latour[6] of its epidemic prevalence in Damascus, and by that of Post,[7] who speaks of it as a common malady at Beyrout. In *Arabia* typhoid is stated by Courbon[8] (with particular reference to Jeddah) to be rare, but to run a rapid course. As regards *India*, not very long ago the dogma passed unchallenged, on the weighty authority of Morehead,[9] that the country enjoyed an absolute immunity from typhoid; and this doctrine was even extended so far as to assert the absence of the disease in the tropics generally. On a former occasion[10] I had brought forward well grounded objections to this peremptory declaration of Morehead; I had shown that typhoid was obviously lying hidden behind the "continued and remittent fevers" of the Anglo-Indian physicians, and that there were clear indications of the disease in the

[1] 'Die Türkei und deren Bewohner,' Wien, 1852, ii, p. 293.

[2] 'Arch. de méd. nav.,' 1868, Juillet, p. 42.

[3] In Leudesdorff's 'Nachrichten,' 1877, xi, p. 6.

[4] 'Edin. Med. and Surg. Journ.,' 1843, April, p. 247.

[5] 'Lond. Med. Gaz.,' 1843-44, i, p. 566.

[6] 'Gaz. méd. de Paris,' 1852, p. 12.

[7] 'New York Med. Recorder,' 1868, June, p. 149.

[8] 'Observ. topogr. et méd. rec. dans un voyage à l'isthmus de Suez,' Paris, 1861, p. 68.

[9] 'Clinical Researches on Diseases in India,' i, p. 307. In the second edition of his work, Morehead corrected this error.

[10] First edition of this work, i, p. 160.

treatises of Annesley, Geddes, and Twining, as well as in the epidemiographical papers by Mouat, Shanks, Kinnis, and others. Shortly after I had made public this conviction of mine, I had the satisfaction of seeing it entirely confirmed. At the present day there is no longer any doubt that typhoid is found in all parts of India, and many observers state that the disease is, in all probability, much more common than the accounts which have hitherto reached us seem to say.

The first perfectly reliable information on the occurrence of typhoid in India is that supplied by Scriven[1] from Burmah. If we are justified, he says, in making a diagnosis of a disease from the *post-mortem* condition taken along with the symptoms, then there is no question that the cases in English soldiers which came under his observation at Rangoon in 1853 were cases of typhoid fever. Belonging to the same period is a second notice from that region by Day,[2] in which mention is made of a "malignant fever," described in such a way as to readily suggest the picture of typhoid. The same is true of the sketch which Murchison[3] has given of a "severe remittent fever" that was prevalent at Prome in 1853. A few years after, Ranking[4] had undoubted cases of typhoid under his observation at Rangoon, and he laid down the dictum that we should have to give this disease an "unreserved admission amongst Indian diseases." Meanwhile there had been trustworthy information about typhoid arriving from Upper India, first from Ewart[5] in Ajmeer (North-West Provinces) and from Scriven[6] in Meerut and Calcutta. Then follow the observations in the same tenour (in their chronological order), by Goodeve,[7] likewise from Calcutta, by Francis[8] from the mountain districts of Bengal, and by Hanbury[9] from Deesa in Gujerât, where the disease was epidemic in 1859. "The silence hitherto observed," says Hanbury, "regarding this disease in the medical reports and returns from this and other countries, is partly explained, no doubt, by the fact already mentioned *that it is one new to medical science*," and I believe I am justified in adding that it is still a sealed book to many practitioners in extra-European countries. In 1862 there came the first accounts of its occurrence in the city of Madras and at many parts throughout the Presidency, by Ranking,[10]

[1] 'Med. Times and Gaz.,' 1854, Jan., p. 79.

[2] Ib., March, p. 231.

[3] 'Edin. Med. and Surg. Journ.,' 1855, April, p. 224.

[4] 'Madras Quart. Journ. of Med. Sc.,' 1861, July, p. 300, 1862, April, p. 284.

[5] 'Indian Annals of Med. Sc.,' 1856, Oct., p. 65.

[6] Ib., 1857, April, p. 511.

[7] Ib., 1859, Jan., p. 141.

[8] Ib., 1859, July, p. 531.

[9] 'Transact. of the Bombay Med. Soc.,' 1861, new ser., vii, p. 144, and 'Statist. Reports of the Army for the year 1861,' iii, p. 335.

[10] 'Madras Quart. Journ. of Med. Sc.,' 1862, July, p. 193.

Cornish,[1] and Johnston,[2] and these were followed by notices of it from the French possession of Pondicherry,[3] from Bangalore,[4] and Secunderabad,[5] and by accounts of typhoid epidemics in Cananore and Calicut[6] in 1870, and Madras[7] in 1878. From the Presidency of Bombay there were accounts of typhoid as early as 1862 by Peet;[8] from the North-West Provinces there are references to similar epidemics at Cawnpore[9] in 1870, Meerut[10] in 1875, and Delhi[11] in 1880. Finally, from the Punjaub, we have the declaration of De Renzy:[12] "My belief is that enteric fever is far more common in the Punjaub than has generally been supposed, that it is constantly mistaken for remittent fever, and that most of the cases under the head of continued fever are really cases of enteric"—a view which now meets with general acceptance among Anglo-Indian physicians, and has found definite expression in the consonant declarations of Bryden,[13] Wall,[14] Francis,[15] Dedrickson,[16] Ewart,[17] Ker Innes,[18] and others. Since 1871 typhoid has been struck out, even in the 'Army Medical Reports,' from the "continued fevers," and introduced under the name of "enteric fever;" but it is undoubted that a correct understanding of this disease, which has been designated as "new to medical science," is still far from being universal among the surgeons of the Indian Service, and the statistical data remain, therefore, much below the reality, as the British military authorities have expressly stated.[19] From 1862 to 1874, 884 cases of sickness from

[1] Ib., 1862, April, p. 291.

[2] Ib., Oct., p. 441.

[3] Huillet, 'Arch. de méd. nav.,' 1868, Févr., p. 85.

[4] Murray, 'Army Reports for the year 1863,' v, p. 516.

[5] Ranking, ib., for 1864, vi, p. 547.

[6] Cornish, in 'Report of the Sanitary Commissioner of Madras for the year 1870.'

[7] Notice in the 'Times of India,' copied into the 'Brit. Med. Journ.,' 1878, Nov., p. 776; and Furnell, 'Med. Times and Gaz.,' 1879, Dec., p. 631.

[8] 'Transact. of the Bombay Med. Soc.,' 1862, new ser., viii.

[9] Moffatt, 'Ind. Annals of Med. Sc.,' 1873, Jan., p. 373.

[10] Ref. in 'Lancet,' 1875, June, p. 805.

[11] Ref. ib., 1880, June, p. 973.

[12] 'Lancet,' 1871, Feb., p. 283.

[13] 'Madras Journ. of Med. Sc.,' 1872, Jan., p. 35, and 'Med. Times and Gaz.,' 1875, Feb., p. 235.

[14] 'Lancet,' 1873, Sept., p. 331.

[15] 'Brit. Med. Journ.,' 1880, Sept., p. 470.

[16] 'Dublin Journ. of Med. Sc.,' 1880, Dec., p. 481.

[17] 'Lancet,' 1880, Feb., p. 291.

[18] 'Brit. Med. Journ.,' 1879, Dec., pp. 993, 1028.

[19] It is a noteworthy fact, in this connexion, that the number of cases reported every year has steadily increased from 1862 to 1874, amounting respectively to 10, 20, 9, 13, 14, 16, 70, 61, 89, 131, 104, 143, and 168. This increase, as the Report says ('Army Reports for 1872,' xiv, p. 144), "is more apparent than real, and seems to be due in a great measure to increased accuracy of diagnosis." The

typhoid came to official notice among the English troops in Bengal (including the North-West Provinces and the Punjaub), among whom were 380 deaths; most of them are reported from the North-West Provinces,—from Lucknow, Meerut, Agra, Allahabad, Umballa, and other places. At a few points the disease appears to be endemic, as at Umballa, where numerous cases of typhoid are observed every year in the regiment stationed there. In 1874, however, there are also many cases of typhoid reported from Lower Bengal. In the Presidency of Bombay cases of typhoid are reported among the British troops in 1871 and 1872; sixty-four cases, of which thirty-nine ended fatally, came under official notice during that period. In the Madras Presidency 165 cases of typhoid among the troops are recorded from 1872 to 1874, with forty-eight deaths.

There is an account by Massy[1] of the occurrence of typhoid in 1865 in *Ceylon*, at Newera Ellia, situated at a height of 6000 feet. In the *East Indian Archipelago*, according to Popp[2] and v. Leent,[3] the disease must be comparatively rare, and it would seem to affect mostly the crews of ships lying in East Indian ports.

From 1865 to 1878, the total number of cases of typhoid or "Zenuwzinkingkoorts" which came under official notice among Indo-Dutch troops stationed in Java and Madura was 662, of which 262 ended fatally.[4] Besides these, a considerable number of fatal cases of febris gastrica and febris biliosa figure in the sick lists, many of which belong, doubtless, to typhoid; so that the above quoted assertions of Popp and v. Leent are hardly borne out by the statistics. In the hospital on the *Andaman Islands*, Brander[5] saw no case of fever from 1877 to 1879,

The statements quoted already as to the occurrence of typhoid in Burmah may be taken along with the reports of French physicians on the disease in *Cochin China*; they

following statement by the surgeon of an artillery division in which typhoid was epidemic in 1874 is characteristic, and important for judging the question of the frequency of the disease in India: "It is believed that a considerable number of diseases returned under other headings were, in reality, cases of enteric fever; but it was considered advisable not to alter the nomenclature in any instance, where any of the pathognomonic symptoms were wanting." The statistical data in the next sentence in the text, showing the enormous mortality of 40 to 50 per cent. of the sick, might of themselves suggest that only a small proportion, and these the severest cases, were correctly diagnosed.

[1] 'Army Reports for the year 1866,' viii, p. 498.

[2] 'Nederl. Tijdschr. voor Geneesk.,' 1859, iii, p. 22.

[3] 'Arch. de méd. nav.,' 1867, Sept., p. 164, 1872, Janv., p. 21.

[4] According to the military health-reports of Beeking, in 'Geneesk. Tijdschr. voor Nederlandsch-Indië,' 1879, viii, pp. 223, 283, 313, ix, p. 83.

[5] 'Edin. Med. Journ.,' 1880, Nov., p. 393.

are unanimous that it is common in Saigon and other French possessions in the basin of the Cambodia in Anam.

Laure[1] had already called attention to the fact that an unusually large number of cases of typhoid had come under notice on board the French ships of war stationed at ports in Further India. At the same time Richaud[2] pointed out the frequent occurrence of the disease among the population of Saigon; he himself had seen, in 1862, nearly 100 cases of typhoid, of which 37 ended fatally, the diagnosis being confirmed by *post-mortem* examination. To the same effect, Gimelle[3] reports from the same place "Fièvre typhoïde assez commune comme partout;" and Danguy des Déserts[4] says the same, adding the remark that the disease there, contrasting with its comparatively rare occurrence in other regions of the tropics, is not only very frequent but also very malignant; there were treated in the hospital in 1864, 107 typhoid cases, of whom 35 died, and in 1870, 135 cases of whom 54 died.[5]

On board English and French ships stationed at Chinese ports as well, or lying in them for a time, a strikingly large number of cases of typhoid have occurred at all times.[6] But in the *Chinese* port towns themselves—we have no information about diseases in the interior—the disease would appear to be anything but rare. Unfortunately the accounts of English practitioners, on which we have chiefly to depend, are, with their "continued fevers," of very little value in estimating the state of matters as regards typhoid; and the recent vague conception of "typho-malarial" fever, which has found its way into the views of practitioners there also, has not altogether helped to elucidate the question. Dudgeon,[7] indeed, speaks of the disease as rare in China; and he adds to this statement the explanation that the Chinese permit no negligence with fæcal matter, which is used at once for manure, so that there is no accumulation of it. But there

[1] 'Hist. méd. de la marine franç.,' Paris, 1864, p. 8.

[2] 'Arch. de méd. nav.,' 1864, Mai, p. 352.

[3] 'Union méd.,' 1869, No. 53, p. 693.

[4] 'Considér. sur l'hygiène de l'Européen en Cochinchine,' Par., 1876, p. 12.

[5] Danguy mentions a severe kind of fever which was prevalent among the Anamese inhabiting the forest region, and known on that account as "fièvre du bois," and he thinks that we have here to do with "typhus;" Brémaud ('Arch. de méd. nav.,' 1879, Dec., p. 423) conjectures, and rightly, that this "fever of the woods" is nothing else than malarial fever.

[6] See Laure, l. c., and the 'Statist. Reports on the Health of the Navy.'

[7] 'Glasgow Med. Journ.,' 1877, April, p. 174.

are facts against him, not only the assertions of Friedel[1] and Rose[2] as to the occurrence of typhoid in Canton and Macao, as well as in Foo-Chow, but also the accounts of epidemic typhoid in 1858 and 1859 among the English troops at Hong Kong,[3] and in 1875 at Shanghai and Pekin, where, it is stated,[4] the disease is never altogether absent. In *Japan*, according to the unanimous opinion of Friedel,[5] Schmid,[6] Simmons,[7] and Wernich,[8] typhoid is prevalent under the same circumstances and with the same frequency as on European soil; the last-named observer designates it as one of the commonest of the infective diseases of Japan.

On the *Australian Continent*, as well as in *Tasmania*, *New Zealand*, *New Caledonia* and other *Islands of the Pacific Ocean*, typhoid takes one of the foremost places among their acute infective diseases in respect of frequency and malignancy.

As early as 1830, Scott[9] and Milligan[10] had observed an epidemic of typhoid among the English troops in Tasmania; the disease likewise showed itself, and became known as "colonial fever" soon after the English began to settle on the coasts of the mainland,[11] and it has been equally frequent in the ships of war on the Australian station.[12] In the account[13] given of the severe epidemic in Melbourne in 1878, attention is called to the steadily increasing frequency of the disease there; whereas the mortality from typhoid in Melbourne had averaged 128 annually from 1869 to 1873, it rose to 226·4 (in a population of 200,000) from 1874 to 1878. Ryley,[14] who makes out a complete parallel between the diffusion of the disease on the Australian Continent and that which has been observed in European countries, expresses the same opinion

[1] 'Beiträge zur Kenntniss des Klimas und der Krankheiten Ost-Asiens,' Berlin, 1863, p. 132.
[2] 'Pacific Med. and Surg. Journ.,' 1862, Oct.
[3] Smart, 'Transact. of the Epidemiol. Soc.,' 1862, i, p. 211.
[4] Leudesdorff, 'Nachrichten,' 1876, x, p. 28.
[5] L. c., p. 34.
[6] 'Madras Journ. of Med. Sc.,' 1870, Sept., p. 232.
[7] 'Amer. Journ. of Med. Sc.,' 1877, April, p. 422.
[8] 'Deutsche med. Wochenschr.,' 1878, p. 100.
[9] 'Transact. of the Prov. Med. and Surg. Assoc.,' 1835, iii, App. xi.
[10] 'Transact. of the Calcutta Med. Soc.,' 1836, viii, App. x.
[11] Bourse, 'Arch. de méd. nav.,' 1867, Mars, p. 164.
[12] Ref. in 'Lancet,' 1875, June, p. 173.
[13] 'Typhoid Fever in Melbourne in 1878' [by W. Thomson], Melb., 1879.
[1] 'Brit. Med. Journ.,' 1880, July, p. 13.

for New Zealand also; from that country there is an earlier account by Thomson,[1] and more recent information by Mackinnon,[2] according to whom typhoid was the commonest and most malignant kind of camp sickness in the campaign of 1864-65 in that colony. In the *Fiji Islands* also, as Ryley states, typhoid is not uncommon. The same applies to the *Hawaiian Islands*, according to Julick,[3] and it applies also, according to the French military surgeons,[4] to *Tahiti*, where the disease is endemic among the Europeans as well as the native population, and has often developed into an epidemic, as in 1847, 1849, and 1853-54. In the same report,[5] mention is made of its frequent occurrence in the *Marquesas Group*; and the authorities are unanimous as to the prominent part that typhoid plays among the acute diseases of *New Caledonia*.[6]

If we now turn, in the course of this geographical survey of typhoid, to *Africa*, we meet first with two considerable foci of the disease in the islands of *Réunion* and *Mauritius*, situated within the tropics. Regarding the former, Dutroulau[7] confirms Oelsner's [8] earlier account of the frequent occurrence of typhoid, adding that the disease becomes epidemic from time to time. For Mauritius also, the statement of Borius [9] that typhoid is endemic in Port Louis and often becomes epidemic, finds full confirmation in the account given by Power.[10] In *Madagascar*, Davidson[11] and Borchgrevink[12] agree that the disease is not at all rarely met with. The *Cape* forms one of its chief seats on the African continent; the definite infirmation about its occurence there dates, indeed, from quite recent times, but we are justified in concluding that it had been one of the chief scourges of the country long before.

[1] 'Brit. and For. Med.-Chir. Review,' 1854, Oct.

[2] 'Army Reports for the year 1865,' vii, p. 407.

[3] 'New York Journ. of Med.,' 1855, March.

[4] See Dutroulau, l. c., p. 57, and notices in 'Arch. de méd. nav.,' 1865, Oct., p. 282.

[5] 'Arch. de méd. nav.,' l. c., p. 297.

[6] De Rochas, 'Essai sur la topogr. hyg. et méd. de la Nouvelle-Calédonie,' Paris, 1860, p. 17; notice in 'Arch. de méd. nav.,' 1866, Janv., p. 22; Charlopin, 'Notes rec. en Calédonie de 1863 à 1867,' Montp., 1868, p. 21; Brun, 'Notes sur quelques cas de fièvre typhoïde à Bourail (Nouv. Calédonie),' Paris, 1879.

[7] L. c., p. 50.

[8] 'Monatsschr. der Berl. geogr. Gesellsch.,' n. s., iv, p. 275.

[9] 'Arch. de méd. nav.,' 1868, Oct., p. 262.

[10] 'Med. Times and Gaz.,' 1870, Jan., p. 120.

[11] Ib., 1868, Dec., p. 646.

[12] 'Norsk Mag. for Laegevidensk,' 1870, p. 247.

In 1863, a considerable epidemic was reported among the Boers in the neighbourhood of Queenstown;[1] there was another epidemic in 1867 at Cape Town and other places in Cape Colony, which reached such enormous dimensions that 5651 cases of typhoid occurred among the 30,000 inhabitants of the town, of whom 360 died.[2] Soon after the diamond fields began to be frequented, typhoid broke out there also, and increased in proportion as the rush of people became greater.[3] In like manner the disease has been so generally diffused through places in Natal, that it now counts among the most prevalent maladies of that region.[4]

Only a few notices relevant to this subject have come to my knowledge for the health-conditions of the *West Coast of Africa*. Moreira[5] says that "typhous fever," distinguishable from typhus by the absence of the exanthem, is not uncommon in Angola. McWilliam,[6] in his account of observations made by him on the "Niger fever" in the unfortunate Niger expedition of 1841, thus sums up the anatomical condition found in eight inspections: "The morbid appearances observed in the intestines are very like those so often found in fatal cases of the typhoid fever of this country."[7] Jenner mentions also the occurrence of typhoid in Sierra Leone; and Chassanoil[8] remarks that typhoid is met with on the Gaboon, rarely among the negroes, but more frequently among the Europeans. How far the general prevalence of the disease on the Guinea Coast may be inferred from this, I am unable to decide. In *Senegambia*, according to the observations published by Defaut[9] for the hospital of Gorée since 1852, typhoid is by no means so rare as earlier writers[10] have stated.

1 Lawson, 'Transact. of the Lond. Epidemiol. Soc.,' 1867, ii, p. 399.

2 Thornton, 'Army Reports for 1867,' ix, p. 381.

3 Egan, 'Med. Times and Gaz.,' 1872, Jan., p. 111, 1873, June, p. 613.

4 Black, ib., 1873, July, p. 107. 1879, Nov., p. 554.

5 'Jornal da socied. das sc. med. de Lisboa,' xv, p. 121.

6 'Medical History of the Expedition to the Niger,' Lond., 1843, p. 146.

7 'Med. Times,' 1853, vi, p. 312.

8 'Arch. de méd. nav.,' 1865, Mai, p. 506.

9 'Hist. clinique de l'hôpital maritime de Gorée,' Paris, 1877, p. 123.

10 Thevenot, 'Traité des malad. des Européens . . . . au Sénégal,' Paris, 1840, p. 171; Dutroulau, l. c., p. 11, who admits, however, that the disease is sometimes found among troops newly arrived, and even in small epidemics; Gauthier, 'Des endémies au Sénégal,' Paris, 1865, p. 17; Chassaniol (l. c.) also states that typhoid is not at all rare in Senegambia among the Europeans.

"Typhoid fever," writes Defaut, "is, one may say, somewhat frequent in Gorée, and in the statistics for 1871, we meet with no fewer than six authenticated cases of the malady. . . . In searching the clinical records of the hospital at Gorée since 1832, we have found that typhoid fever occurs every year to something like the same extent;" and he adds that the diagnosis had always been confirmed in fatal cases by the *post-mortem* examination.

Maclean[1] has seen typhoid in *Ascension;* and in the *Canary Islands,* according to Busto y Blanco,[2] epidemics of typhoid are met with from time to time.

Like many other tropical or subtropical countries, *Algiers* has long enjoyed the reputation of being little visited by typhoid; but the experiences of the last ten or twenty years have materially shaken the belief in the immunity of the country from this disease. Antonini[3] and Bertherand[4] had already, in 1845-50, observed several epidemics of typhoid, large and small, not only among the French troops, but more or less diffused among the natives. Confirming this, and justifying us in concluding that the disease is relatively frequent in Algiers, there are later accounts by Masse[5] of an epidemic in Aumale in 1865, by Frisson[6] of one the same year in Ténés, by Longuet[7] of one in 1878 in Sid-bel-Abbés, as well as communications by Arnould and Kelsch,[8] Sorel,[9] and others.

In the small garrison of Ténés, as Frisson informs us, 113 persons died of typhoid from 1843 to 1863; and in the epidemic of 1866, there were 39 cases and 20 deaths among a force of 360 men.

From *Tunis* there is information by Ferrini[10] of the not unfrequently observed occurrence of typhoid as an epidemic in autumn. Courbon[11] has seen the disease also in *Abyssinia,*

[1] 'Lancet,' 1880, May, p. 859.
[2] 'Topogr. med. de las islas Canarias,' Sevilla, 1864.
[3] 'Rec. de mém. de méd. milit.,' 1845, vol. l, 50.
[4] 'Médecine et hyg. des Arabes,' Paris, 1855, p. 529.
[5] 'Rec. de mém. de méd. milit.,' 1866, Avril, p. 293.
[6] Ib., 1867, Juin, p. 433.
[7] Ib., 1879, Nov. et Decbr., p. 561.
[8] Ib., 1868, Janv., p. 17.
[9] 'Union méd.,' 1880, Oct., p. 689.
[10] 'Saggio sul clima e sulle precipue malattie . . . . di Tunisi,' Milano, 1860, p. 187.
[11] L. c., p. 30.

and particularly often during the rainy season; and in *Egypt* it would appear to be by no means so rare as Griesinger[1] and Isambert[2] have concluded from their observations —extending, however, only over a short period in Cairo. According to the account of Penay (published by Griesinger),[3] the disease is prevalent in Khartoum at the time of the rains, and is moreover very widely spread and very malignant. Of its occurrence in Alexandria, Cerf-Mayer[4] says: "La fièvre typhoïde, avec les mêmes symptômes qu'elle offre en Europe, a aussi sa place dans le cadre nosologique;" and, according to Vauvray,[5] the deaths from typhoid in Port Said amount to 6 per cent. (?) of the total mortality.

In the same general diffusion and with equal frequency as in Europe, typhoid is prevalent on the soil of *North America*, and its area of distribution extends, as Drake[6] and Bartlett[7] show, from the Gulf of Mexico to Hudson's Bay Territory. In *Greenland*, where typhoid has appeared as an epidemic several times during the last thirty years, it has been chiefly the northern[8] districts that have suffered, seldom the interior.[9] Also in *Newfoundland*, epidemics of typhoid have been observed several times by Anderson;[10] and, according to Blaschke, they have occurred in New Archangel,[11] or as it is now called, *Alaska* [Sitka]. In the United States, the disease is most prevalent in *Maine* and other *New England States;* next to these, most frequently in the States of *New York*, *Pennsylvania*, *Ohio*, *Michigan*, and the *Prairie States;* scarcely less so in the Central and Western States,[12] such as

[1] 'Archiv für physiol. Hlkde,' 1858, xii, p. 4. Among 200 cases of "typhous sickness," only 15 were well-marked cases of ileo-typhus.

[2] 'Gaz. méd. de Paris,' 1857, p. 233.

[3] L. c., p. 374. No information about the morbid anatomy.

[4] 'Arch. de méd. nav.,' 1869, Mai, p. 327.

[5] Ib., 1873, Sept., p. 180.

[6] 'Treatise on the Principal Diseases of the Interior Valley of North America,' Philad., 1854, ii, p. 358.

[7] 'History . . . of the Fevers of the United States,' Philad., 1852, p. 107.

[8] 'Sundheds-Kolleg. Forhandl.,' 1849, p. 22, 1854, p. 41, 1857, p. 297.

[9] Lange, 'Bemerkn. om Grönlands Sygdomsforhold,' Kjöbenh., 1864, p. 32.

[10] 'Dobell's Reports,' i, p. 365, ii, p. 198.

[11] 'Topogr. med. portus Novi-Archangelcensis,' Petropol., 1842, p. 64.

[12] "It is the common fever of the Eastern States," says Bartlett, "it prevails also more or less extensively in the Middle and Western States." The first

Virginia,[1] *Kentucky*, *North Carolina*,[2] *Tennessee*,[3] and *Missouri*.[4] In the Southern States, also, in *South Carolina*,[5] *Georgia*,[6] *Alabama*,[7] *Mississippi*,[8] *Louisiana*,[9] and *Texas*, typhoid is by no means rare. For the States west of the Rocky Mountains, there are accounts of the prevalence of the disease in *Utah*,[10] *Oregon*,[11] *California*,[12] and *Alaska* (as above).

The disastrous prevalence of typhoid among the Federal troops in the War of Secession in 1862-63 furnishes a striking proof of the enormous extent to which the disease may develop on the soil of the United States when the circumstances are particularly unfavorable, and in the Central and Southern States as much as in the Northern.[13] In those two years there occurred in the Atlantic region (including the coast belt between the Alleghany Mountains and the sea, from New England down to Florida), in a force of 460,000 men (in

accounts of the occurrence of typhoid in the New England States were published by Jackson ('Report founded on Cases of Typhoid Fever, &c.,' Boston, 1838), after observations in 1821–35 in the Massachusetts General Hospital, and by Hale ('Remarks on the Pathology of the Typhoid Fever of New England'). Later accounts of its general diffusion may be found, for New Jersey, in the 'Transact. of the N. J. State Med. Soc.,' 1861, p. 37; for Pennsylvania, in the 'Transact. of the Penns. State Med. Soc.,' 1859, 1864, 1866, 1871, 1872, 1873, pp. 137, 153, 156; for Michigan, by Bailey, in the 'Transact. of the Amer. Med. Assoc.,' 1859, xii; and for Indiana, by Byford, in the 'Amer. Journ. of Med. Sc.,' 1851, Jan., p. 61.

[1] Bartlett, l. c.; Reeves, 'Pract. Treatise on Enteric Fever,' Philad., 1859; Bland, in 'Transact. of the West-Virginia State Med. Soc.,' 1871.

[2] McKee, in 'Fenner's South. Med. Reports,' ii, p. 405; Dickson, 'Transact. of the Amer. Med. Assoc.,' 1860, xiii.

[3] Bartlett, l. c.; Grant, 'Amer. Journ. of Med. Sc.,' 1853, July, p. 94.

[4] 'Notice in 'Transact. of the Amer. Med. Assoc.,' 1855, viii.

[5] Gibbs, Gaston, and others, in 'Proc. S. Carol. Med. Assoc.,' 1854, Columb., 1855.

[6] Bartlett, l. c.; Posey, in 'Transact. of the Amer. Med. Assoc.,' 1857, x.

[7] Drake, l. c., p. 412; Clarke and Lewis, in 'New Orleans Med. Journ.,' 1847, July, pp. 9, 31.

[8] Bartlett, l. c.

[9] Gibbs, in 'Fenner's South. Med. Rep.,' ii, p. 185, and numerous epidemiographical notices in the 'New Orleans Med. Journ.'

[10] Bartholow, 'Amer. Journ. of Med. Sc.,' 1860, April, p. 324.

[11] Glisan, ib., 1865, Jan., p. 78.

[12] For the general diffusion of typhoid in California, see Blake, ib., 1852, July, p. 53; Stillman, 'Edin. Med. and Surg. Journ.,' 1852, Oct., p. 283; King, 'Amer. Journ. of Med. Sc.,' 1853, April, p. 389; Praslow, 'Der Staat Californien, &c.,' p. 36; Oatman, 'Transact. of the California State Med. Soc.,' 1857.

[13] The data are taken from the preliminary report on the 'Medical and Surgical History of the Rebellion' (Circular No. 6 of the War Department), Philad., 1865, p. 109.

round numbers) 29,666 cases of typhoid, of which 7092 had a fatal issue. In the central region (the Mississippi Valley between the Alleghanies and the Rocky Mountains), in a force of 403,000 men there were 23,530 cases of typhoid, 8970 of them fatal. In the Pacific region, again (Oregon and California), out of a force of 15,408 men (who were, however, little in action) there were only 155 cases with thirteen deaths. The rate of sickness therefore was, in the Atlantic region 64·2 per thousand, and in the Central region 58·4, while the mortality was 15·4 and 22·3 respectively. In this calculation no account is taken of 12,093 cases of "typho-malarial fever" with 489 deaths, which figure in the reports from the Atlantic region.

In *Mexico* typhoid occurs as frequently as in the United States, and chiefly on the tableland (the so-called Tierra fria)[1] and on the eastern slope of the Andes (on the Tierra templada, at elevations of 1000 to 1500 metres);[2] on the other hand, it is rare on the coast belt. Heinemann says[3] that he had not seen a single case of typhoid at Vera Cruz during a period of six years, but it is clear from the papers of Naphegyi[4] and Saillant[5] that the disease has been observed there from time to time; according to the report of the latter, eight cases of typhoid had been treated in the Marine Hospital of Vera Cruz in 1864 and 1865. Liddell, of Panama,[6] and Schwalbe, of Costa Rica,[7] agree in the opinion that typhoid is no stranger to the littoral of *Central America*.

As to the disease in the islands of the *West Indies*, the data of the various authorities are very contradictory; but so much, at least, may be concluded from them in general, that typhoid is by no means so seldom met with there as is usually assumed; and that, on some islands at least, it takes a not unimportant place in the list of infective diseases.

One of the first trustworthy pieces of information as to the occurrence of typhoid in the West Indies occurs in the work of Levacher,[8] who

[1] Jourdanet, 'Gaz. méd. de Paris,' 1865, pp. 193, 251; Coindet, 'Rec. de mém., de méd. milit.,' 1864, Mai; Wuillot, 'Presse méd. belge,' 1866, No. 40.

[2] Poncet, 'Rec. de mém. de méd. milit.,' 1863, Févr., p. 91 (account of the endemic prevalence of the disease in Orizaba).

[3] 'Virchow's Archiv,' 1873, vol. 58, p. 161.

[4] 'New York Journ. of Med.,' 1855, May.

[5] 'Notes méd. rec. à l'hôpital de la marine de Vera Cruz,' Paris, 1869, p. 11.

[6] 'New York Journ. of Med.,' 1852, March, p. 255.

[7] 'Archiv für klin. Med.,' 1875, xv, p. 344.

[8] 'Guide méd. des Antilles,' Paris, 1840, p. 57.

saw the disease in *Sta. Lucia*; he begins his account of it with the words: "There is no country, so far as I know, where the stages and crises of the fever follow more punctually or can be better observed." In *Martinique*, according to Dutroulau[1] and Rufz,[2] typhoid is rare, and occurs only in the case of new arrivals, among whom Rufz saw the disease epidemic in 1841. Batby-Berquin[3] expresses himself to the same effect for *Guadeloupe*. On the other hand, Carpentin[4] mentions that typhoid occurred as an epidemic in 1867 at Camp Jacob, situated at a height of some 500 metres, not only among the newly arrived troops, but also among the natives and the acclimatised civil population; that from 1857 to 1869, 248 cases had occurred in the military hospital alone (out of a small force of about 500 men); and that the disease must be regarded as endemic there. That was also the opinion of Brassac,[5] arrived at ten years before, from his experience as surgeon-in-chief from 1858 to 1860. Of the epidemic and sporadic occurrence of typhoid in *Barbadoes*, there are recent accounts by Jackson[6] and Fergusson.[7] From *Trinidad* we have information of a severe epidemic of typhoid in 1866-67 by Bakewell[8] and Stone,[9] the latter remarking that the practitioners told him they had never seen (or perhaps recognised?) the disease there before. It appears, also, that the *Greater Antilles* have not escaped typhoid, from the accounts of severe epidemics of it in *Cuba* in 1853-54, and in *San Domingo* in 1871-72 by Dupont,[10] and Llenas;[11] in Cuba, where epidemics of typhoid would appear to be by no means uncommon, the number of cases in those two years was 2488, of whom 459 died. The epidemic prevalence of typhoid in the *Bermudas* is mentioned in the official reports[12] for 1866, when it occurred among the civil population as well as among the British troops, and for 1869, when it was confined to a single regiment of 710 men (forty-three cases and nine deaths).

In *Cayenne* typhoid has several times attained to epidemic diffusion, as in the winter of 1852-53, when some 500 cases were observed among the convicts.[13] From *Surinam*, also,

[1] L. c., p. 35.

[2] 'Arch. de méd. nav.,' 1869, Aug., p. 135.

[3] 'Notes sur quelques malad. observées à la Guadeloupe,' Paris, 1873, p. 42.

[4] 'Étude hyg. et méd. du Camp-Jacob.,' Paris, 1873, p. 41.

[5] 'Arch. de méd. nav.,' 1865, Mars, p. 227.

[6] 'Bost. Med. and Surg. Journ.,' 1867, July, p. 447.

[7] 'Lancet,' 1880, April, p. 540.

[8] 'Med. Times and Gaz.,' 1868, April, p. 457.

[9] Ib., 1868, Feb., p. 200, and 'Brit. and For. Med.-Chir. Rev.,' 1868, July, p. 237.

[10] 'Notes et observ. sur la côte orientale d'Amerique,' Montp., 1868, p. 53, based on the data of Piña y Pinuela ('Topogr. méd. de la Isla de Cuba').

[11] 'Contribution à l'histoire des maladies de S. Dominigue,' Paris, 1874, p. 37.

[12] 'Brit. Army Reports' for 1866, viii, p. 63, and for 1869, xi, p. 75.

[13] Dutroulau, l. c., p. 20.

there is information which points to its somewhat frequent occurrence there.[1] As to its frequency in *Brazil*, and more particularly in Rio, the only place from which there is any information on the subject, there are very conflicting opinions among the practitioners, which appear to me to have arisen, not so much out of the facts themselves, as out of the more or less incorrect interpretation of them. Some physicians are of opinion that typhoid had been prevalent there in former times; while others, such as Sigaud,[2] believe that it came first in 1836 by importation into Rio by a Spanish vessel from the Canaries, admitting, however, that the disease has never become extinct since then, and has, on several occasions, as in 1842, reached to epidemic diffusion. On the other hand, Lallemant[3] states that only sporadic cases of typhoid came under his notice from 1845 to 1848, and these were in fresh arrivals from Europe; Bourel-Roncière mentions the opinion, prevalent there at the time, that a "mephitic condition" had developed in the city in consequence of the canalisation-works in progress in 1863 and 1864, and that this condition, joined to a "malarious soil," had given rise to an epidemic of typhoid;[4] and Homem[5] asserts that the disease since 1870 has been more common in the city than formerly, and that the earlier cases of typhoid were mostly errors of diagnosis. It appears, at any rate, to be well established that the disease now figures not inconspicuously in the statistics of sickness at Rio. In the *River Plate States* typhoid counts among the prevalent infective diseases.[6] The same is true of *Chili;* earlier observers (Pöppig,[7] Brandin,[8] and Gilliss[9]) had called attention to the frequent occurrence of "typhous fever," known there under the name of "chavalongo;" and more recently the French

[1] V. Leent, 'Arch. de méd. nav.,' 1881, Févr., p. 99.

[2] 'Du climat et des maladies du Brésil,' Paris, 1844, pp. 192, 251.

[3] 'Casper's Wochenschr. für die Hlkde.,' 1845, p. 470, 1849, p. 545.

[4] 'Arch. de méd. nav.,' 1873, Mars, p. 204.

[5] 'Estudio clinico sobre as febres do Rio de Janeiro.' (Abstract in the 'Arch. de méd. nav.,' 1879, Jan., p. 50.)

[6] Dupont, l. c., p. 17; Féris, 'Arch. de méd. nav.,' 1879, Oct., p. 247.

[7] 'Clarus und Radius Beiträge zur pract. Heilkd.,' 1834, i, p. 528.

[8] 'De la influencia de los differentes climas sobre el hombre,' Lima, 1826.

[9] 'U. S. Naval Astronom. Exped. to the Southern Hemisphere,' Washington, 1855.

naval surgeons, who had opportunities of seeing many cases of that disease at Valparaiso, have come to the conclusion that the disease is really typhoid.[1] Ullersperger[2] gives an account of an epidemic of typhoid which occurred at Santiago and other places in Chili in 1864, using as his authority the annals published by the University of Santiago. I am unable to decide from independent evidence how far we may trust the statement of Tschudi[3] that "typhus" is generally diffused along the littoral of *Peru*, as well as in the Puna and Sierra regions, and how far this "typhus" corresponds to typhoid; I have met with only one notice relating to the point from Callao, by Fournier,[4] according to whom typhoid appears to be common there. From Guayaquil (*Ecuador*) we have an account by Sigaud,[5] following Hauvel, a surgeon of the French navy, of a severe epidemic of typhoid which broke out among the beseigers during the siege of the town in 1834, and afterwards spread to the town itself, and in the end to the ships lying in the harbour. More recently, Duplouy[6] has mentioned the disease as one occurring frequently there. Finally, I shall direct attention to the interesting statement of Metcalfe,[7] that sporadic cases of typhoid occur from time to time on *Norfolk Island*, a small point of land situated in the Pacific, between New Caledonia and New Zealand, in lat. 29° 3′ S. and long. 167° 58′ E.

§ 154. An Ubiquitous Disease; Comparative Immunity of the Tropics.

The sketch of the geographical distribution of typhoid given above in outline, justifies the designation of an *ubiquitous* disease which I gave to it at the beginning of the chapter; it proves, in fact, that the incidence of the disease is as wide as the globe, and it disposes of the

[1] Notice in 'Arch. de méd. nav.,' 1864, Août, p. 102.
[2] 'Virchow's Archiv,' 1869, vol. 48, p. 501.
[3] 'Oest. med. Wochenschr.,' 1846, No. 12.
[4] 'Arch. de méd. nav.,' 1874, Sept., p. 254.
[5] L. c., p. 197.
[6] 'Arch. de méd. nav.,' 1864, Oct., p. 281.
[7] 'Brit. Med. Journ.,' 1880, Nov., p. 740.

opinion, which had been widely accepted until quite recent times, that the *tropics* enjoy a complete immunity from it. Typhoid is certainly rarer in lower latitudes than in higher, and especially rare in the tropics; but the facts which have lately reached us concerning the diffusion of the disease in India, make it doubtful whether this relative immunity is in reality as great in other tropical regions, as it seems to be at present. At all events, in the facts hitherto established of the occurrence of typhoid throughout the whole of India, in Burmah, Cochin China, Réunion, Mauritius, Madagascar, Tahiti, Sennegambia; in the frequently observed epidemic outbreaks of it in the West Indies and the Bermudas, in Cayenne, Brazil, and other countries; and in the remarkably frequent cases of typhoid sickness among the crews of ships in harbours in the tropics (whereof more anon)—we have in all this, together with the general diffusion of the disease in temperate and high latitudes, evidence that the *climate*, or, in other words, the physical nature of the atmosphere characteristic of the particular region, does not *in and by itself* exert a determining influence on the occurrence of typhoid, and that the reason of the comparative rarity of the disease in the tropics must be attributed, therefore, to other circumstances.

§ 155. Relation to the Seasons.

That it is not the high *temperature*, giving to the tropical climate its essential character, which accounts for this relative immunity of the tropics from typhoid, may be inferred from the fact that a high state of the thermometer in general not only does not preclude the prevalence of the disease, but even favours it to a certain extent, and that the point of greatest prevalence falls mostly in the warm or hot season. In order to make clear this question as to the relative amount of typhoid in the several *seasons*, I subjoin in the first place, the results of a statistical inquiry into the circumstances in question in various parts of the world.

From this statistical conspectus of the amount of sickness in the several seasons of the year, we obtain the interesting

fact that, at by far the larger number of places of observation, the maximum of cases falls in late summer and autumn and the minimum in spring; that in some regions, the summer, and in others the winter, takes a lower place than the spring; and that only in Munich and Prague does the number of cases (or of deaths) in winter exceed that of autumn, while at Stuttgart, in Sweden, and at Hamburg, the two are almost equal. Taking the amount of sickness in spring as unity, we arrive at the following ratios.

Table of the Ratios of Typhoid in Autumn, Winter and Summer[1] *reckoning the Spring Sickness as* 1.

Place.	Autumn.	Winter.	Summer.
Copenhagen	4·9	2·1	2·9
Drammen	3·4	2·2	1·5
Lausanne	3·3	1·9	1·9
London	3·2	1·7	2·2
Paris	2·9	1·6	1·8
Massachusetts	2·8	1·3	1·6
Leipzig	2·7	1·7	2·1
Christiania	2·4	2·2	1·3
Boston	2·4	1·2	1·6
Frankfurt, o. M.	2·2	1·3	1·5
Berlin	2·0	1·2	1·4
Nassau	2·0	1·6	1·3
Geneva	1·9	1·7	1·0
Chemnitz	1·9	1·4	1·8
Basel	1·7	1·3	1·3
Glasgow	1·7	0·9	0·9
Pittsburg	1·5	1·0	0·9
Breslau	1·5	1·2	1·3
Sweden	1·2	1·2	1·1
Hamburg	1·2	1·3	0·9
Stuttgart	1·2	1·3	1·0
Munich	0·7	1·3	0·7
Prague	0·7	1·3	0·7

The result obtained from these tables, that the amount of the sickness touches its highest point in autumn, is fully borne out by the facts as to the season of greatest prevalence of typhoid in many other localities;

[1] Under the term "summer" I understand the months June, July, and August; under autumn September, October, and November; under winter December, January, and February; and under spring March, April, and May. The only exception to this in the next table (p. 644) is in the case of Sweden, where the figures in the official statistics are given for the quarters (Jan.—March and so on) and not for each month.

Table of Admissions or Deaths from Typhoid in the several Months and Seasons.

Place.	Period.	Admissions *or* Deaths.	Months.												Seasons.			
			June.	July.	August.	September.	October.	November.	December.	January.	February.	March.	April.	May.	Summer.	Autumn.	Winter.	Spring.
Sweden[1]	1858–77	Admissions	—	—	—	—	—	—	—	—	—	—	—	—	44,750	49,334	51,573	42,354
Christiania[2]	1845–64	,,	154	281	402	393	437	768	602	517	335	283	196	182	837	1598	1454	661
Drammen[3]	1861–67	,,	46	100	149	180	253	251	202	141	92	88	56	55	295	684	435	199
Copenhagen[4]	1842–58	,,	162	254	428	588	526	317	328	195	105	103	92	100	844	1431	628	295
Hamburg[5]	1873–80	Deaths	82	82	122	116	147	127	158	146	149	125	90	102	286	390	453	317
Berlin[6]	1854–79	,,	850	1159	1616	1879	1965	1540	1184	997	919	854	921	910	3625	5384	3100	2685
Breslau[7]	1863–78	,,	187	215	244	287	267	220	202	197	192	192	164	154	646	774	591	510
Leipzig[8]	1851–65	Admissions	64	98	137	135	144	99	76	100	60	54	44	41	299	378	236	139
Chemnitz[9]	1837–75	,,	171	208	303	300	245	185	241	148	166	121	112	154	682	730	555	387
Prague[10]	1874–76	,,	78	90	69	79	76	84	115	191	122	119	106	110	237	239	428	335
Nassau[11]	1818–59	,,	1118	1406	1742	2093	2350	2207	1946	1850	1584	1428	1060	848	4266	6650	5380	3336
Frankfurt o. M.[12]	1863–80	Deaths	52	74	91	106	113	93	76	60	58	50	50	43	217	315	194	143
Stuttgart[13]	1852–77	,,	69	76	83	87	88	108	122	106	84	90	73	66	228	283	312	229
Bavaria[14]	1857–70	,,	—	—	—	—	—	—	—	—	—	—	—	—	10,758	11,648	12,722	12,037
Munich[15]	1852–68	,,	408	377	379	365	363	425	619	718	783	699	548	444	1164	1153	2120	1691
	1873–79	,,																
Neuchatel[16] Lausanne	1835–52	,,	57	72	95	125	159	92	88	81	49	52	25	38	224	376	218	115
Geneva[17]	1838–45	,,	—	—	—	—	—	—	—	—	—	—	—	—	105	213	180	109
Basel[18]	1824–73	,,	169	186	202	237	237	236	193	192	143	137	121	160	557	710	528	418
London[19]	1848–62	Admissions	163	220	333	361	377	334	222	197	122	136	89	103	716	1072	541	328
Glasgow[20]	1871–79	,,	12	15	30	43	36	31	20	23	18	29	18	17	57	110	61	64
Paris[21]	1867–78	Deaths	205	289	511	559	522	565	429	259	240	192	205	176	1005	1646	928	573
Massachusetts[22]	1846–48	,,	—	—	—	—	—	—	—	—	—	—	—	—	671	1182	544	429
Boston[23]	1840–47	Admissions	30	47	86	92	98	60	48	39	43	40	21	41	163	250	130	102
Pittsburg[24]	1873–77	Deaths	27	32	65	64	90	65	52	53	37	43	44	53	124	219	142	140

[1] From the official returns of cases of typhoid in 'Sundhet's-Kollegiums Berättelse om Medicinalverket i Sverige.'

[2] From the data of Conradi as to the number of admissions into the Rigs-hospital ('Norsk Mag. for Laegevidensk.,' 1860, p. 14, 1862, p. 714, 1864, p. 902, 1865, p. 1020).

[3] From Holst, l. c., giving the total number of cases in Drammen and vicinity.

[4] Admissions into the General Hospital, according to Trier, l. c., p. 21.

[5] Number of deaths, according to reports of the Medical Inspector.

[6] Mortality due to typhoid, from official reports; see also Virchow, 'Gesammelte Abhandl. aus dem Gebiete der öffentl. Med.,' Berlin, 1879, ii, pp. 435, 469; and the statistical year-books for the City of Berlin published by Böckh, Nos. iv—vi.

[7] Total mortality from typhoid in the city, according to Jacobi, 'Beitr. zur med. Klimatol. und Statistik der Stadt Breslau,' Bresl., 1879, p. 53.

[8] Admissions into the Julius Hospital, according to Thomas ('Archiv der Heilkd.,' 1866, p. 400).

[9] Admissions into the hospital, according to Flinzer ('Statist. Mittheil.,' parts 2 and 3, Chemn., 1875, p. 43, 1877, p. 53).

[10] Popper, 'Prager Viertelj. für Heilkd.,' 1878, iii, p. 4, according to the cases registered in the city.

[11] From official returns of sickness in 'Med. Jahrb. des Herzogthums Nassau,' No. 15-16, p. 591, and No. 19-20, p. 223.

[12] Mortality returns in 'Statist. Mittheil. vom Civilstand u. s. w. der Stadt Frankfurt a. M.'

[13] Mortality throughout the population, according to Cless, 'Württ. med. Corrspdzbl.,' 1878, p. 234.

[14] From official returns in 'Bayr. med. Intelligenzbl.,' 1872 p. 337.

[15] From official returns of the mortality in the city; conf. Pettenkoffer, 'Zeitschr. für Biologie,' 1868, iv, p. 7, and 'Bayr. med. Intelligenzbl.,' 1873-79.

[16] Cornaz, 'Étud. statist. sur la fièvre typhoïde,' Anvers, 1854.

[17] Marc d'Espine, l. c.

[18] Hagenbuch, 'Journ. für Kinderhlkde,' 1875, p. 46.

[19] Admissions into the London Fever Hospital, according to Murchison, l. c., and Tweedie, 'Lancet,' 1860, Feb.

[20] Admissions into the Fever Hospital, according to Macphail, 'Glasg. Med. Journ.,' 1879, Oct., p. 257.

[21] From the reports of Besnier ('Union méd.,' 1867-79) giving the deaths from typhoid in the Paris hospitals. The figures are wanting for Jan.—Sept., 1870, and Oct.—Dec., 1871.

[22] The mortality throughout the State, according to Curtis, 'Transact. of the Amer. Med. Assoc.,' ii, p. 487.

[23] Admissions into the Lowell Hospital, according to Bartlett, l. c., p. 119.

[24] Snively, 'Transact. of the Pennsylvania State Med. Soc.,' 1878, p. 285.

for example, in Schwerin,[1] Bremen,[2] and other districts of Germany; in Iceland, where, out of six epidemics, the disease reached its height four times in late summer and autumn; in Malta (Marston); in Italy, where the highest point in the larger number of typhoid epidemics fell in autumn; at the Cape, where, out of four epidemics, three fell in summer and autumn and one in winter; in Greenland (Lange) and Newfoundland (Anderson); in Pennsylvania,[3] California (Praslow), and Oregon (Glisan).

All the more noteworthy is the circumstance that, in tropical and subtropical regions, it is chiefly the hot months that form the typhoid season.

Of three epidmics in Algiers, for which the time is given, two were in summer and autumn and one in summer; Arnould and Kelsch state that the maximum falls there in summer; and that applies also to Tunis (Ferrini), Japan (Friedel), and the River Plate States (Dupont). All observers in India (Bryden, Hanbury, and others) are agreed that most cases of typhoid occur in the hot season, and, next to that, in the season corresponding to our autumn; in Bengal, according to Don, 75 per cent. of all the cases observed fell within the periods named. In Cochin China, typhoid is prevalent in July (Danguy), in New Caledonia[4] at the time of greatest heats, in the Bermudas in summer and autumn, and in Cuba mostly in the months from June to November (Dupont). In Guadeloupe, on the other hand, according to several years' observations by Brassac, the disease occurs chiefly in autumn and the beginning of winter; in Rio, also, as Homem states, typhoid is mostly seen in autumn (March to June).

§ 156. Relation to the Temperature.

This association of the disease, in its development and diffusion, with particular seasons, points to some influence exerted by certain meteorological conditions which make themselves felt at one season or another, according to the locality, the kind of soil, or other peculiarities proper to the climate of the region. Certain *states of the weather*, therefore, would principally determine the pathogenesis. To decide precisely what the grounds are of this assumption

[1] Brückner, 'Archiv für wissensch. Med.,' 1867, iii, p. 160.

[2] Lübstorff, 'Beitr. zur Kenntniss des öffentl. Gesundheitszustandes der Stadt Lübeck,' Lüb., 1862, p. 17.

[3] According to numerous statements in the 'Transact. of the Pennsylvania State Med. Soc.' Variau (l. c., 1873, p. 137), in his account of the epidemic in the late summer and autumn of 1872 in Crawford County, speaks of it as "the endemic fever of the season."

[4] Notice in 'Arch. de méd. nav.,' 1866, Janv., p. 22.

—or, to put it more generally, what the meteorological factors are which exert that influence on the amount of sickness—is not so easy from a comparison between the weather-conditions in various localities visited by the disease at various seasons, and the amount of sickness or mortality in them respectively, as from a comparison of the amount of sickness in one and the same place at different times, regard being had to the prevalent weather-conditions. All investigators hitherto have followed the latter line of investigation, without, however, coming to a harmonious conclusion. That no special importance in this connexion can be ascribed to the temperature of the air—high or low—*by itself*, follows from the fact that the acme of the disease falls variously in various regions within higher latitudes, either in autumn or in winter; while, in the tropics, it falls mostly at the time of the greatest heats. From a table which I have prepared of numerous epidemics, I elicit the fact that typhoid is almost as common in cool as in warm summers, at the cold as at the warm period of autumn, in mild as in severe winters. The following tabular survey of the mortality from typhoid in Berlin in the several months of the year, from 1871 to 1878, side by side with the temperature, the rainfall, and the height of the sub-soil water at the time, may be useful for judging of this and other questions to follow, as it is based upon observations very carefully collected over a very considerable period.[1]

Table of Deaths from Typhoid in Berlin.

	1871	1872	1873	1874	1875	1876	1877	1878	Mean.
Number of deaths . . .	47	81	61	22	43	49	47	20	—
Mean temperature (Reaumur)	-3·99	0·61	3·28	2·45	1·40	-1·66	2·49	1·49	-0·65[2]
Rainfall (in tenths of an inch)	14·96	19·78	10·92	17.12	39·07	8·67	27·83	18·58	17·02[3]
Sub-soil water . . .	1·94	1·81	1·90	1·66	1·37	1·81	1·59	1·62	—
Number of deaths . . .	31	44	60	41	65	42	40	17	—
Mean temperature (Reaumur)	-1·00	1·39	0·11	1·77	-2·79	1·90	2·57	3·29	0·74
Rainfall (in tenths of an inch)	23·21	8·00	5·40	7·15	9·55	38·12	54·93	6·55	16·72
Sub-soil water . . .	2·02	1·84	1·91	1·71	1·52	1·91	1·78	1·73	—

[1] The data are taken from the statistical year-book for the City of Berlin issued by Herr Böckh. [2] Mean of 25 years. [3] Mean of 15 years.

Table of Deaths from Typhoid in Berlin—(continued).

		1871	1872	1873	1874	1875	1876	1877	1878	Mean.
March	Number of deaths	35	32	85	70	58	34	33	21	—
	Mean temperature (Reaumur)	5·09	5·00	3·81	3·79	0·96	3·98	2·56	3·50	2·68
	Rainfall (in tenths of an inch)	8·58	14·63	19·05	28·07	12·32	59·50	17·35	43·25	15·08
	Sub-soil water	2·42	1·91	1·93	1·75	1·57	2·33	2·06	1·87	—
April	Number of deaths	52	40	110	47	41	22	39	15	—
	Mean temperature (Reaumur)	5·89	8·69	6·00	8·49	6·71	7·85	5·56	8·33	6·76
	Rainfall (in tenths of an inch)	27·65	22·85	6·40	13·45	10·42	14·07	8·10	16·75	19·98
	Sub-soil water	2·43	1·99	1·98	1·85	1·76	2·53	2·21	2·04	—
May	Number of deaths	46	48	90	33	46	46	20	18	—
	Mean temperature (Reaumur)	8·33	12·01	9·10	8·71	11·37	8·16	9·03	11·43	10·71
	Rainfall (in tenths of an inch)	16·18	23·30	23·30	20·50	31·27	5·97	15·03	20·08	24·70
	Sub-soil water	2·29	1·97	1·87	1·87	1·80	2·22	2·09	1·97	—
June	Number of deaths	30	50	54	33	46	48	21	22	—
	Mean temperature (Reaumur)	11·32	14·03	14·48	14·01	15·37	14·78	15·83	14·11	13·88
	Rainfall (in tenths of an inch)	61·05	18·25	21·57	20·37	28·02	28·10	16·18	30·38	32·55
	Sub-soil water	2·15	1·84	1·75	1·74	1·63	1·96	1·90	1·82	—
July	Number of deaths	33	99	49	55	75	57	34	26	—
	Mean temperature (Reaumur)	15·15	16·37	16·18	17·10	15·65	15·69	15·59	13·94	15·05
	Rainfall (in tenths of an inch)	33·85	10·74	40·92	12·37	20·15	20·72	21·10	30·88	37·15
	Sub-soil water	2·09	1·69	1·64	1·52	1·56	1·75	1·69	1·67	—
August	Number of deaths	72	128	68	82	148	77	87	40	—
	Mean temperature (Reaumur)	15·12	13·95	12·52	13·55	16·57	15·33	15·16	15·16	14·48
	Rainfall (in tenths of an inch)	10·30	10·48	19·02	22·17	14·00	14·12	52·60	33·33	26·13
	Sub-soil water	1·96	1·57	1·56	1·38	1·41	1·55	1·49	1·55	—
Septmbr.	Number of deaths	108	134	108	92	162	73	93	46	—
	Mean temperature (Reaumur)	11·49	12·75	11·24	13·72	11·94	11·07	9·66	12·75	11·62
	Rainfall (in tenths of an inch)	17·78	16·25	19·85	8·70	10·92	31·25	21·85	11·30	16·12
	Sub-soil water	1·83	1·48	1·49	1·29	1·32	1·43	1·50	1·49	—
October	Number of deaths	100	287	73	86	114	66	107	38	—
	Mean temperature (Reaumur)	5·51	8·91	8·80	9·35	5·52	9·49	6·75	9·21	7·75
	Rainfall (in tenths of an inch)	16·53	27·25	13·80	6·22	56·72	7·60	16·60	9·80	17·36
	Sub-soil water	1·75	1·44	1·48	1·23	1·29	1·37	1·47	1·44	—
Novmbr.	Number of deaths	72	174	55	82	68	66	53	37	—
	Mean temperature (Reaumur)	1·75	5·93	4·55	2·56	2·23	1·66	6·02	3·89	2·84
	Rainfall (in tenths of an inch)	9·55	36·08	18·04	9·55	31·40	26·27	13·03	9·28	18·36
	Sub-soil water	1·73	1·56	1·50	1·82	1·46	1·40	1·47	1·43	—
Decmbr.	Number of deaths	106	91	46	54	73	43	38	26	—
	Mean temperature (Reaumur)	−1·35	2·10	2·77	0·09	−0·63	0·90	1·70	0·80	0·53
	Rainfall (in tenths of an inch)	14·43	19·13	21·30	24·80	14·77	17·00	15·58	16·43	19·29
	Sub-soil water	1·77	1·75	1·61	1·27	1·65	1·48	1·56	1·46	—

If we extract from this table a few facts specially indicating the relation of the amount of sickness to the height of the temperature, we

find that, in spring, the severest mortality (285 deaths) was in 1873, with a temperature 0·43° R. below the mean, that the lowest in spring (54 deaths) was in 1878 with a temperature of 1·02° R. above the mean, and that, of intermediate death-rates, that of 1871 (133 deaths) corresponded to a temperature 0·29° R. below, and that of 1872 (116 deaths) to 1·83° R. above the mean. The severest summer endemics, those of 1872 and 1875 (282 and 269 deaths) had a temperature respectively of 0·31° and 1·39° above the mean of twenty-five years; on the other hand, with almost the same temperatures in 1876 and 1877 (0·79° and 1·06° above the mean) there were only about half the number of deaths (182 and 142 respectively).

Just as we find for spring and summer no constant relation between the degree of temperature and the amount of mortality from typhoid,[1] equally little is the assertion borne out—for Berlin—that the maxima of the autumnal epidemics correspond to years with high summer temperature.

The severest autumnal epidemic in the period from 1871 to 1878 falls in the autumn of 1872 (596 deaths), the temperature having been 1·79° R. above the mean, while it had been 0·31 above the mean in summer. On the other hand, in 1875, when the highest summer temperature of the eight years was reached (1·39 above the mean), there were only 344 deaths from typhoid in autumn (with a temperature 0·84° below the mean), or rather more than one half the number in 1872. Next to 1875, the highest summer temperature was in 1877 (1·06° above the mean) and in 1876 (0·79° above); those years far exceeded 1872, and yet the deaths from typhoid were no more than 253 and 205, or about one-third the number in 1872.

§ 157. Relation to the Rainfall.

Greater stress as a cause of typhoid is laid on the *atmospheric moisture*, or on the *rainfall*, than on the atmospheric temperature; and attention has been particularly called to the frequency of its epidemic prevalence in dry years, especially in an autumn following a hot summer with little rain.

Cless, in his history of the epidemics of mucous fever in Stuttgart,[2] has shown that a hot and dry summer had preceded the appearance of the disease in autumn on several occasions, and that the outbreak of

[1] I would especially point out that the relation in question does not come out differently when the calculation is made for shorter periods, or for single months. The table supplies many evidences of this; I may mention particularly the month of August in 1872 and in 1875, and the month of July in 1872 and in 1873.

[2] L. c., p. 108.

the epidemic then, coincided with the setting-in of wet weather; but he showed no desire to carry his conclusions further.[1] Other observers arrived at their conclusions much more quickly; among the rest, Murchison,[2] who found in the English experiences of 1846 and 1860 a confirmation of the opinion that typhoid occurred with especial severity after those summers which were characterised by their drought and heat, while it was remarkably slight in summers and autumns which were cold and wet. Still more decided was the declaration of Zülzer,[3] on the ground of his Berlin observations of 1863-66: "The colder and wetter a season is, the less is the mortality [from typhoid] in the season following; the smaller the amount of rain (in proportion to the temperature), or the warmer the season has been, the more severe will typhoid be in the next three months." Trier[4] also concluded, from inquiries covering the period from 1842 to 1858, that, in Copenhagen, dry and warm summer weather favoured the development of the disease, although he had to admit that there were many exceptions to this rule. Socin[5] in like manner inferred from his inquiries into typhoid at Basel from 1864 to 1869, that unusual drought preceding would encourage the development of typhoid epidemics; but he leaves it open whether it was the drought or the subsequent rains (both of them as affecting the soil) that supplied the actual favouring cause.

On the other hand, Buhl,[6] who had meanwhile been carrying out his acute inquiries as to the influence of the height of the subsoil water on the development of typhoid, became convinced "that in Munich it was not to the meteorological precipitations that we should assign the determining cause of the amount and intensity of typhoid." Seidel,[7] indeed, taking up the inquiry in turn, thought that he had made out "that in Munich, during any month with a greater rainfall than the average of the season, there will more probably be a decline in the number of typhoid patients below the average of the particular month, than a rise above the average, and *vice versâ*;" but Pettenkofer[8] showed that this view is justified only under certain circumstances. The doctrine

[1] "It is certainly not difficult," he remarks with profound truth (p. 110), "to bring the whole group of antecedent influences into relation with the epidemic, and to interpret it accordingly; so that at last we think that we have proved in the clearest manner that this epidemic must necessarily have originated at this particular time. But he who thus accounts for everything, accounts in the end for nothing."

[2] L. c., p. 411.

[3] 'Beiträge zur Aetiologie . . . . der typhoiden Krankheiten,' Berlin, 1870, p. 37.

[4] L. c. [5] L. c., p. 14. [5] 'Zeitschr. für Biologie,' 1865, i, p. 7.

[6] Ib., 1866, ii, p. 169. [7] Ib., 1868, iv, p. 13.

of the influence of meteorological precipitations on the production of typhoid underwent further criticism, by means of a series of direct observations on the state of the weather at the time of the outbreak and prevalence of epidemics in various parts of Europe.

Thus Maclagan,[1] among others, confirming the opinion that had been hazarded by Socin, showed from his experiences in Dundee and its vicinity, that it was not the drought preceding the epidemic that constituted the essential etiological factor, but the subsequent rains. Marston[2] also had adopted that opinion on the strength of facts observed in Malta; and the same view was arrived at by Fleischmann[3] after an experience of many years at the Bavarian fortress of Kaisheim; by Hjaltelin, who says that in Iceland the disease becomes epidemic just towards the end of a wet summer; and by Thomas,[4] who shows that, in Leipzig, typhoid occurs much more abundantly in wet years than in dry. Others again uttered a warning against overrating the dryness of the air as an etiological factor in the question before us; among the rest, Colin,[5] who says, on the evidence of various facts that had come to his notice: "These facts prove how much exaggeration there is in always blaming the drought."

The following data, from the Berlin statistics of typhoid and associated conditions of the weather, serve to estimate the significance which is to be ascribed to meteorological precipitations (independently of the temperature or in connexion with it). Arranging the years in the order of the amount of rainfall, proceeding from maximum to minimum, we get the following table.

Table of the Rainfall and Typhoid Mortality at Berlin.

Year.	Rainfall (in tenths of an inch).	Deaths from typhoid.
1877	280·18	612
1875	278·61	939
1876	271·39	623
1871	253·77	732
1878	246·61	326
1872	226·74	1208
1873	219·58	859
1874	190·37	697

[1] 'Edin. Med. Journ.,' 1867, Oct., p. 297, and 'Lancet,' 1868, July, p. 116.
[2] 'Brit. Army Reports,' 1861, iii, p. 492.
[3] 'Bayr. med. Intelligenzbl.,' 1877, No. 1.
[4] 'Archiv der Heilkde,' 1866, p. 407.
[5] 'De la fièvre typhoïde dans l'armée,' Paris, 1878, p. 143.

The smallest mortality (326) and the largest (1208) both occur in years (1878 and 1872) with moderate rainfall (the mean for fifteen years at Berlin being 260·64‴); the second largest (939) falls in a very wet year; but there is no constant relation discoverable between the volume of the annual rainfall and the height of the death-rate. The following table shows the relation between the death-rate in autumn and the kind of weather in the preceding summer.

Table of the Autumnal Death-rate from Typhoid at Berlin, and of the Weather during the previous Summer.

Year.	No. of deaths.	Summer		Autumnal	
		Temp.	Rainfall.	Temp.	Rainfall.
1871	280	13·86 R.	115·20‴	6·25 R.	43·86‴
1878	121	14·60	94·59	8·62	30·38
1877	253	15·53	89·28	7·48	51·48
1873	236	14·39	81·51	8·20	51·70
1876	205	15·26	62·94	7·40	65·12
1875	344	15·86	62·17	6·56	99·04
1874	260	14·88	54·91	8·54	22·47
1872	596	14·78	39·47	9·20	79·58

The severest autumnal epidemic (1872), under a very high temperature and copious rains, followed on a very dry and moderately warm summer; the next most severe autumnal epidemic (1875) falls in a cool and very wet autumn, which had been preceded by a warm and moderately damp summer; the third in order of severity (1871), under a very cool temperature (*i.e.* 1·25° R. below the mean) and moderate rains, followed an excessively wet (nearly 30‴ above the mean) and cool summer; and the circumstances of the fourth severe epidemic (1877) are similar. The smallest mortality (1878) falls in a warm and dry autumn, which had been preceded by a moderately warm and very wet summer. Lastly, I shall advert to the circumstances in question as they obtained in the two severe winter epidemics of 1871-72 (229 deaths) and 1872-73 (218 deaths); the winter of 1871-72 was cold and moderately dry, and was preceded by a dry and cold autumn; the winter of 1872-73 was unusually warm and dry, but it was preceded, on the other hand, by a very warm and damp autumn.

As regards Berlin, there is no definite relation whatsoever to be made out between the height of the death-rate from typhoid and the states of the weather; and the inquiries at other places or in other countries have led to the same result.

Franque[1] thus sums up the experience of Nassau in this matter from 1818 to 1853: "We can ascribe to meteorological conditions hardly any direct influence on this form of disease." Larsen[2] says that the uniform course which the disease in Norway follows in its diffusion every year cannot be brought into definite connexion with states of the weather. Stedman,[3] for Boston, and many other American physicians, express themselves to the same effect.

But I consider the question of an influence exerted by the weather, and particularly by the rainfall, on the development and spread of typhoid, as answered in the negative only in so far as relates to a *direct* or *constant* influence of this etiological factor. Inasmuch as the spread of the disease stands probably in a certain relation to processes in the soil, and above all to the amount and to the movements of moisture therein, we may conjecture *a priori* that a more or less abundant saturation of the soil by atmospheric precipitations will, under particular circumstances of locality, determine the course of the endemic or epidemic sometimes towards one side sometimes towards the other—in the one case, under certain local conditions, copious rains would be favorable to the development of the disease, in the other case a scanty rainfall.

§ 158. Associated with Certain Conditions of Locality.

In its *mode of occurrence and diffusion in space* typhoid shows a peculiarity which distinguishes it very characteristically from all other acute infective diseases, and stamps it as a morbid process intimately bound up in its origin with *conditions of locality*. Typhoid occurs sporadically unusually often; in many other instances it is prevalent as an endemic; or, finally, it attains to an epidemic diffusion of longer or shorter duration. But these typhoid endemics or epidemics are always confined within local limits; they never spread over considerable tracts of country; the disease has never assumed the character of a wide-spread epidemic or of a pandemic. And if there have been, from time to time, epidemic outbreaks of it simultaneously at many places near to or remote

[1] 'Nass. med. Jahrb.,' 1854, pt. 12–13, p. 884.

[2] L. c., p. 97.

[3] 'Boston Med. and Surg. Journ.,' 1879, Oct., p. 500.

from one another, these have not had any intimate connexion, as in the case of wide-spread epidemics of cholera, scarlatina, typhus, or yellow fever; but it is generally in them a matter of local conditions making themselves felt at a number of points simultaneously, but independently of each other.

"The epidemics of typhoid fever," says Besnier,[1] are *local* epidemics, and their exacerbations are absolutely *local* also. While typhoid was raging in an intense form at Lyons not long ago, I took the trouble to ascertain that its type at Paris was simple and benign. While it is to-day raging at Paris severely and extensively, it continues rare and benign elsewhere, even in the same country at the same moment, often even within the same town, and under a perfectly uniform constitution of the atmosphere."

Typhoid in this respect connects most closely with malarial diseases, without, however, attaining the same wide diffusion. This strictly local character of the disease comes out with quite peculiar prominence in the fact that it very often remains confined to single quarters of a town, to single streets or groups of houses, not unfrequently even to single buildings, to institutions for the reception of certain classes, such as barracks, educational institutes, boarding-houses, and prisons, or even to private houses, the whole neighbourhood round the narrowly-circumscribed focus of disease enjoying a complete immunity.

An interesting example of such a focus of disease, within narrow local limits, is furnished by the epidemic of 1843 at Torgau, in which the sickness was confined almost exclusively to the principal barracks situated at the highest point of the town, and to the quarter adjoining.[2] Examples of the development of circumscribed foci of disease in public institutions occur in the literature in large numbers.[3] In the

[1] 'L'Union méd.,' 1876, No. 131, p. 683.

[2] Koeppe, 'Der Abdominaltyphus in Torgau im Jahre 1843,' Eilenburg, 1847.

[3] See the accounts in the more recent literature, relating to garrison epidemics, by the following: Buxbaum ('Zeitschr. für Biol.,' 1870, vi, p. 1) for Freising in 1865 and 1868; Widall ('Rec. de mém. de méd. milit.,' 1870, Dec., p. 449) for Maubeuge in 1869; Schmiedt ('Deutsche milit.-ärztl. Ztg.,' 1875, p. 78) for Blankenburg a. H. in 1873; Pfeiffer ('Jen. Zeitschr. für Naturw. und Med.,' 1868, iv, p. 21) for Weimar in 1867; Apoiger ('Bayr. ärztl. Intelligenzbl.,' 1877, Nos. 10—15) for Burghausen in 1875; Page ('Brit. Army Report for 1874, xv, p. 301) for Kildare in 1874; Simon ('Notice sur une petite épidémie de fièvre typh. à Cherbourg,' Par., 1874) for Cherbourg in 1874; Caradec ('Gaz. des hôpit.,' 1880, No. 144) for Brest in 1879; and the accounts in the work by Colin

same class we include the often observed outbreaks on board ship, especially in the ports of tropical countries, accounts of which have been received from the Dutch East Indian possessions,[1] from the port of Calcutta,[2] from Cochin China and from China,[3] from Tahiti,[4] from the coast of Senegambia,[5] from the West Indies,[6] Surinam,[7] and other places. In many of the cases of this kind the disease developed itself among the crew during the voyage.

§ 159. Occurs in Elevated and Low Situations equally.

This mode of occurrence and spreading of typhoid indicates that the development of the disease is bound up with definite local conditions, of the spot or circumscribed area affected endemically or epidemically by the sickness; it indicates that certain factors situated in the *soil*, or in the *hygienic circumstances* of those localities, furnish material conditions for the origin and commencement, as well as the continuance of typhoid—called endemic or epidemic according to its duration.

It is clear, from a glance at the distribution of area of typhoid, that *elevation* and *configuration of the ground* in themselves have no influence on the occurrence of the disease. Observations made at the most diverse points of the globe—in Thuringia,[8] Upper Bavaria,[9] and other parts of Germany, in Switzerland, in many parts of France,[10] and England, in

which I have quoted several times. See also reports by Fleischmann ('Bayr. ärztl. Intelligenzbl.,' 1877, Nos. 1—3) on typhoid in the penal establishment at Kaisheim, Bavaria; by Zuckschwerdt ('Die Typhusepidemie im Waisenhause zu Halle a. d. S. im J. 1871,' Halle, 1872) on the epidemic in the Halle orphanage; by Brown ('Philad. Med. Times,' 1875, Nov., p. 80) on epidemics in boarding-houses at Mansfield in the autumn of 1874, and Burlington in the winter of 1874-75.

[1] 'Arch. de méd. nav.,' 1867, Sept., p. 164.

[2] Dedrickson, 'Dublin Journ. of Med. Sc.,' 1880, Dec., p. 481.

[3] Laure, l. c., p. 8.

[4] Notice in 'Arch. de méd. nav.,' 1865, Oct., p. 282.

[5] Thevenot, l. c.

[6] Hayne, 'Lancet,' 1875, May, p. 616.

[7] Popp, 'Nederl. Tijdschr. voor Geneesk,' 1859, iii, p. 217.

[8] See Pfeiffer, 'Zeitschr. für Epidemiol.,' 1868, i, Nos. 1 and 2; Thomas, ib., 1870, ii, p. 153; Lothholz, 'Beitr. zur Actiol. des Typhus,' Jena, 1866, p. 22.

[9] Wibmer, 'Bayr. ärztl. Intelligenzbl.,' 1858, p. 327.

[10] Gaultier de Claubry, 'Mém. de l'Acad. de Méd.,' 1843, x, p. 3; Kergaradec, ib., xxvi, p. 222, and xxvii, p. 116.

Sicily,[1] in Bradford County,[2] and other counties of Pennsylvania, in North[3] and South[4] Carolina, in California,[5] in Mexico, and elsewhere—go to prove that the disease is met with as frequently on the coast as in the interior, on level as on undulating surfaces, within basins as on table-lands, in valleys as on mountain ridges. Some observers, indeed, emphasize the fact that, where there are slight differences of level, the disease mostly affects the lowest streets or quarters of towns, whether large or small, and that the elevated localities, on the other hand, are less affected or even quite exempted; but in many other instances, those differences between the amount of sickness at low and at high lying points have not been manifestated, or the relation has even been exactly the inverse.

In Berlin, on the observations of five years (1863—1867), the elevation of the several areas of typhoid had no recognisable relation to the mortality among the residents in them.[6] In Leipzig, it is true, the parts of the town lying lowest are worse off in general than the more elevated quarters; but there are considerable exceptions to this rule, notably the eastern suburb, which is much less visited by the disease than the southern suburb situated on the highest ground of the city.[7] In Basel, as Socin has found,[8] the course of typhoid has proceeded quite uniformly in all parts of the town, whether they were situated high or low, no quarter enjoying any striking preference in this respect. In Zürich, as Wegelin remarks,[9] the quarters lying highest very often become the seats of typhoid epidemics. In the epidemics of 1873-74 among the garrison of Brussels, the barracks situated on high ground were affected as much as, and even more than, those at a low elevation;[10] and this fact came out still more strikingly during the severe epidemic there in 1868-69, in which the high-lying parts of the city were the head-quarters of the disease.[11] In the epidemic of 1843 at Torgau,

[1] Merletta, 'Istoria della febbre epid. comparsa in Palermo,' Palermo, 1828.
[2] Holmes, 'Transact. of the Pennsylv. State Med. Soc.,' 1859.
[3] Dickson, l. c.
[4] Gaston, 'Proc. of the South Carolina Med. Soc.,' 1854.
[5] Oatman, 'Transact. of the California State Med. Soc.,' 1857.
[6] Zülzer, l. c., p. 72. This fact is confirmed for the years 1877-78 by Skreczka ('Viertelj. für gerichtl. Med.,' 1879, Jan., p. 152).
[7] Thomas, 'Arch. der Hlkde.,' 1866, p. 406.
[8] L. c., p. 47.
[9] 'Der Typhus im Canton Zürich,' Zürich, 1854, p. 21.
[10] Gys, 'Arch. méd. belges,' 1874, April, p. 217.
[11] De Change, ib., 1868, Juin; notice in 'Presse méd. belge,' 1869, No. 9; V. d. Corput, ib., No. 36; Mascart, ib., 1870, No. 26.

which has been mentioned several times, the disease remained restricted almost entirely to the principal barracks situated at the highest point of the town (59 feet above the level of the Elbe), and to a small section of the streets running at an equal elevation, while the lower quarters suffered little, and the very lowest escaped altogether.[1] In like manner, during the epidemic at Lausanne in 1841, it was precisely the parts of the town situated lowest that enjoyed complete immunity.[2] Bartlett[3] mentions the following interesting fact for Lowndesborough, Alab., on the authority of Wooten. In the neighbourhood of the town, some three miles distant from the Alabama river, there is a hilly elevation about six miles in length, on which the owners of plantations in the adjoining flats have built their residences. Malarial fever is very extensively prevalent all round about, but typhoid rarely occurs; whereas, on this hilly belt, which is almost entirely free from malaria, typhoid occurs so often that many planters prefer to remain on their plantations, and take malarial fever into the bargain, rather than expose themselves to an attack of typhoid.

§ 160. Relation to the Physical Characters of the Soil.

As to the relation between typhoid production and the *geological characters of the soil*, or the *kind of rock* in the locality, it is self-evident that the question can only arise in so far as concerns the *physical characters* of the soil dependent thereon—its permeability to moisture and air. And in this sense we have to take all those observations which go to show that the disease is met with oftener on the more recent formations than on the older.

Magne[4] has made inquiries into this matter, using as his materials the epidemiological reports sent in to the Paris Academy of Medicine from 1841 to 1863 (with the exception of 1858); and he comes to the following conclusion:—Of the 757 epidemics reported, 564 have occurred in 224 arrondissements resting on the more recent formations (alluvium, diluvium, and tertiary); 64 in 54 arrondissements on primary or transition rock; 129 in 77 arrondissements whose soil belongs partly to the older and partly to the recent formations. There have been accordingly 251 epidemics per 100 arrondissements on recent formations, 167 per 100 in those with a mixed soil, and 118 per 100 on the older rocks. There are no accounts at all of typhoid epidemics from 47 arrondissements of the first group (recent), 27 of the second (mixed), and 20 of the third (older); so that these in their order have escaped the disease in the ratio of 20 : 35 : 37. It follows that it is not

[1] Koeppe, l. c., p. 25.
[2] De la Harpe, 'Gaz. Suisse de méd.,' 1844, No. 2.
[3] L. c., p. 117.
[4] 'Bull. de l'Acad. de méd.,' 1865-66, xxxi, p. 94.

the extent of the arrondissement or the density of its population that determines the frequency or rarity of typhoid in it, from the fact that there is one epidemic for each district with 56,832 hectares and 37,081 inhabitants in the first group, with 94,918 hectares and 62,514 inhabitants in the second, and with 115,467 hectares and 26,206 inhabitants in the third.

Even if this conclusion can by no means be taken to hold good absolutely, owing to the extremely incomplete character of the epidemiological returns known to have been made to the Academy—a fact that appears at once in the comparatively small number of epidemics of typhoid announced during twenty-two years—yet the conclusion is still deserving of consideration; and it has been corroborated in several subsequent inquiries relating to smaller areas.[1] But it is still the permeability of the soil which we shall have to look to as the proper determining factor, having regard not only to the lower soil, but even more, to the upper, or, in other words, laying the whole stress upon the *penetrability of the ground by air and moisture*, and upon its *hygroscopic character*. We must bear in mind that a more or less considerable layer of mineral or vegetable detritus on the oldest rock imparts to the soil the same physical characters which the alluvial, diluvial, or other recent formations possess.

§ 161. Associated with Fluctuations in the Sub-soil Water.

The number of inquiries hitherto conducted with exactness in this direction is still much too small to yield moderately safe conclusions on *the influence of the saturation of the ground upon the production of typhoid*. So far as we may form an opinion, neither absolute dryness nor absolute dampness of the soil appears to be decisive for one side or the other, inasmuch as typhoid is as often prevalent on wet as upon dry ground. The proper determining factor would be the *fluctuations in the amount of moisture in the soil*, the

[1] Thus Beaufort ('Bull. gén. de thérap.,' 1868, lxxiv, p. 506) remarks of the endemic typhoid of the Depart. Indre, that, in the centre of the department where granite and liassic limestone form the basis, the disease is much more widely spread over the limestone than over the primary formation, and that it occurs mostly on the latter where there is an argillaceous stratum over the granite.

changing from dampness to dryness; and, in order to judge of that, the movements of the sub-soil water afford the sole reliable criterion. It is well known that Buhl, stimulated by the pioneering inquiries of Pettenkofer, has earned the credit of having first tested the relation between the amount of typhoid and the highth of the sub-soil water, his observations having been made at Munich. He arrived at a law which he formulates[1] as follows: *Between the fluctuations of the sub-soil water and the amount and severity of typhoid there is an unmistakable connexion: in this wise, that the total of the cases of sickness and death from typhoid falls with the rise of the sub-soil water, and rises with the fall of it; that the level reached by the disease is not in proportion, however, to the then level of the sub-soil water, but only to the variation in it on each occasion; or, in other words, that it is not the high or low level of the sub-soil water that it is decisive, but only the range of fluctuation.* This law, worked out first for Munich, and for the period from 1856 to 1864, has been verified there for subsequent years,[2] and has found a complete confirmation in the observations instituted on the same plan at other places.

An agreement with the Munich observations, although not an absolute one, was made out by Schiefferdecker[3] in stating the relation of the disease to the fluctuation of the sub-soil water at Königsberg (but in 1867, typhoid was very prevalent notwithstanding a great rise of the sub-soil water). Pfeiffer[4] made out that the outbreak at Weimar in 1867 was preceded by a great fall of the ground-water; and the same has been noticed in the severe epidemics at Paris in 1876,[5] Tübingen in 1877,[6] and elsewhere. Socin[7] found that the law held good, according to his inquiries, at Basel, although in some years it was less definite than in others; and Pribram and Popper[8] are entirely in agreement with him according to their Prague experiences from 1873 to 1876. With them, also, is Jacoby,[9] for Breslau, who emphasises the fact that

[1] 'Zeitschr. für Biologie,' 1865, i, p. 12.

[2] See Pettenkofer, ib., 1868, iv, p. 1; Seitz, 'Bayr. ärztl. Intelligenzbl.,' 1873, No. 52.

[3] 'Berl. klin. Wochenschr.,' 1868, p. 137.

[4] 'Jen. Zeitschr. für Med.,' 1868, iv, p. 24.

[5] 'Longuet, 'Union med.,' 1877, No. 95; Vallin, 'Gaz. des hôpit.,' 1877, No. 61; Bourdon, 'Lancet,' 1877, Jan., p. 45.

[6] Schmidt, 'Die Typhus-Epidemie in Füsilierbataillon zu Tübingen,' Tübing., 1880.

[7] L. c., p. 23.

[8] L. c., p. 14.

[9] 'Beitr. zür med. Klimatologie und Statistik der Stadt Breslau,' Bresl., 1879, p. 56.

it is not the absolute height or depth of the sub-soil water that is the determining factor there, but solely the range of its fluctuations. In like manner Virchow, for Berlin, says:[1] "We have a certain number of cases of typhoid at all times. The number rises when the sub-soil water falls, and decreases when it rises. Every year, at the time of the lowest level of the sub-soil water, we have a small epidemic."[2]

Certainly other observers, within their own circles of work, have not found evidence to support the principle developed by Buhl. Some of these objections fall to the ground, for the reason that no measurements of the sub-soil water had been taken, a decision having been arrived at solely from the amount of rainfall or the hight of the stream by the water-mark—neither of which furnishes a thoroughly reliable measure of the level of the sub-soil water or of the extent of its fluctuations. In the case of other objections, there has been an erroneous interpretation of the law, in that the standard that has been used to determine the conditions in question has not been the range of fluctuation, but solely the high or low level of the sub-soil water. Still other objections, such as those of Schiefferdecker for the epidemic of 1867 at Königsberg, and of Biermer[3] for the epidemic of 1872 at Zürich, have really had the effect of showing that the law does not hold good equally for all times and places. That this should be so we can understand on reflecting that we are dealing with a factor of disease whose potency may be materially modified by many circumstances of the locality, particularly of its soil; but, above all, on reflecting that fluctuations in the sub-soil water, as well as saturated states of the ground in general, are not in *every case* the decisive thing for an outbreak of an epidemic of typhoid; for the disease has occurred in localities where there can be hardly any question of an influence exerted on the surface soil by fluctuations in the sub-soil water, owing to the water-

[1] 'Generalber. über die Arbeiten der Städtl. Deputation für die Untersuchung der auf Canalisation und Abfuhr bezügl. Fragen,' Berlin, 1874. Reprinted in his 'Gesammelte Abhandl. a. d. Geb. der öffentl. Med.,' Berl., 1879, ii, p. 337.

[2] In the table on pp. 647, 648, the height of the sub-soil water at Berlin is given for the several months of the years from 1871 to 1878, and these relations may easily be followed in it.

[3] 'Ueber Entstehung und Verbreitung des Abdominaltyphus,' Leipzig, 1873 p. 18.

bearing stratum being very deeply placed; and typhoid has been epidemic, as we shall see, under circumstances in which influences of the soil have been excluded altogether. All such facts do not serve to overthrow Buhl's law actually, but merely to prove that there are circumstances, even if we cannot for the present define them, in which its applicability is not incontestable.

§ 162. Relation to Neglected Hygiene.

In passing to consider the question how far *neglected hygiene* has an influence on the occurrence and spread of typhoid, I shall first call attention to a result that I have arrived at from comparing the typhoid mortality in the several counties of England,[1] as shown in the table on pp. 625-26. It is at once obvious that the differences which present themselves here are not to be explained by the conditions of climate or of soil. The highest mortalities fall mostly in the districts that are the great seats of trade and manufacture, or in the principal mining districts of the country; whereas the lowest mortalities are met with in those counties which contain for the most part only small country towns, and in which agriculture and cattle-rearing form the chief means of subsistence of the population. In this I seem to find a further confirmation of the opinion which has been often expressed, that *typhoid occurs more frequently in the great centres of commerce and industry than in country districts.* It may readily be surmised that in typhoid, as in many other acute infective diseases, the defects of hygiene which are commonly felt most severely in large and densely-inhabited towns, or in country districts with a crowded and poor population, furnish an important etiological factor for the production and spread of the disease. This surmise finds confirmation not only in

[1] I do not forget that absolute trustworthiness cannot be claimed, as regards the death-rate figures, for the statistical materials that are here furnished to us; but I may assume that the same sources of error occur throughout the several data, and the details may therefore be used in comparison one with another.

the observations which have been made at many places, that typhoid is prevalent in localities unfavorably situated as regards hygiene, and that the disease breaks out often and over a great extent in a time of general misery such as a time of war,[1] but also, and even more, in the fact, to be afterwards discussed, that a diminution in the amount of typhoid goes hand in hand with improvements in hygiene and with the sanitation of towns.

There is no reason, then, to doubt the importance of this etiological factor for the production of typhus; but when we compare, on the one hand, the circumstances under which this disease breaks out and runs its course, with those of typhus and relapsing fever on the other, we shall find that there are material differences between them. Whereas the latter diseases, as we have seen, are associated almost exclusively with the injurious effects of miserable living—with overcrowded, filthy, and badly-ventilated lanes and houses, with the abodes of poverty and misery—and almost never occur except among the veritable proletariat, and only exceptionally among the masses in better circumstances; typhoid, on the other hand, displays in its outbreaks and its continuance a much greater degree of independence of these influences. The disease has on many occasions been prevalent in the fashionable quarters of a town not less than in the quarters inhabited by the proletariat, in the comfortably-built houses of the well-to-do residents than in the dens of poverty, among the so-called higher ranks than among the working classes; and even the highest positions have afforded no protection from the disease, as events in the royal families of England and Portugal have shown.[2]

"This much is made out," says Cless,[3] speaking of the epidemic of 1835-36 at Stuttgart, "that it was the upper classes which were mostly affected at the beginning of the epidemic, and the lower classes not until later." In Köppe's account of the epidemic at Torgau in 1843, we are reminded[4] that "the disease was located in only a small part of the town, and almost exclusively in the part inhabited by well-to-do

[1] Compare the facts relating to typhoid in the American War of Secession, given at p. 637.

[2] Murchison, l. c.

[3] 'Geschichte der Schleimfieber-Epidemieen,' p. 41.

[4] L. c., p. 25.

citizens and the higher military personages. . . . . In the order of rank, there were one hundred and fifty-three patients belonging to the corps of officers, the civil and military officials, and the merchant class, seventy-two belonging to the tradesmen class, fifty-six to the artisan class, and twenty to the labouring class;" it was not until October that the proletariat was attacked, and then only to a slight extent. In the epidemic of 1868 at Langensalza, the disease was almost confined to the quarter inhabited by prosperous citizens, only a few cases occurring in the overcrowded quarters of the poor.[1] The severe epidemic of 1868-69 in Brussels, according to the unanimous opinion of the authorities, had been prevalent first, and always to the greatest extent, in the richest and most fashionable quarters of the city.[2] De la Harpe makes a similar statement for the epidemic of 1841 at Lausanne; and, for Basel, Socin[3] says: "No part of the town seemed to have any decided preference of immunity in consideration of the sparseness or density of its population." Edmonstone,[4] in his account of the epidemic of 1817 at Newcastle, says: "The fever has been very little known among those classes of the inhabitants, and in those parts of the town, in which infectious fever [*i.e.* typhus] generally rages most extensively, namely, the labouring poor, and the low, filthy, confined situations." In the Windsor epidemic of 1858, according to Murchison,[5] it was particularly the rich and middle classes that were attacked; and it was the same in the Croydon epidemic of 1852, where "the victims were not among the poor, but among the well-to-do and the best of the artisan class in the town."[6] Ewart's statement,[7] based on his observations in Calcutta, is: "It prevailed in the palaces of the rich and well-to-do, and in the huts and hovels of the poor classes." Bartlett[8] sums up the United States' experiences in these words: "In regard to the action of putrid substances, and to the influence of scanty and unhealthy food, it is sufficient, perhaps, to say that there is no satisfactory evidence of their operation in giving rise to the disease." Folsom,[9] referring to observations made in the New England States, says: "Typhoid fever is by no means a disease of the filthiest towns or of the filthiest parts of towns;" and, in the account of the epidemic of 1876 in Philadelphia, it is stated:[10] "The first point which impresses us, in glancing over the table of ward returns, is the uniform dissemination of the disease throughout the entire city. Instead of confining itself to the Delaware front and certain densely-crowded and notoriously insanitary sections, as heretofore, it has existed to an alarming extent in every quarter, and in some of the healthiest districts."

[1] Seyfarth, 'Zeitschr. für Epidemiologie,' 1869, i, No. 4.

[2] See p. .

[3] L. c., p. 47.

[4] 'Edin. Med. and Surg. Journ.,' 1817, Jan., p. 79.

[5] 'Edin. Med. Journ.,' 1859, Aug., p. 149.

[6] Murchison, 'Continued Fevers.'

[7] L. c., p. 293. [8] L. c., p. 127. [9] L. c., p. 229.

[10] 'Transact. of the Pennsylvania State Med. Soc.,' 1877, p. 690.

§ 163. Special Relation to Faulty Sewerage.

The somewhat uniform diffusion of typhoid among people in all circles of society and under the most various conditions of living, contrasting with the more circumscribed occurrence of typhus, relapsing fever, plague, and the like, within the social strata least favorably situated, is a consequence, as we shall see, partly of the particular mode of diffusion which the morbid poison takes, whereby it is brought more or less uniformly to all classes, and partly also of the somewhat special affinity which the disease in its origin shows to *one* offence against hygiene which not unfrequently obtains just among those conditions of living and residence which seem to be the best—I mean the pathogenetic influence associated with the inadequate removal of animal exuviæ, and particularly of human excrement, in other words, with the accumulation of fæcal matter in cesspits, drains, and the like, or with the percolation of those matters into a porous soil to which air and moisture have access.

There are few points in the etiology of typhoid on which there is so much agreement in the opinions of observers, as on the influence exerted by these nuisances on the development of epidemics or endemics of typhoid, or on the occurrence of isolated cases; although there is still much difference of opinion as to the import of the connexion which subsists between the cause of the disease and the morbid condition ensuing. The best evidence of this connexion has been obtained, naturally, from experiences in small, narrowly circumscribed and more easily surveyed fields of observation—in single houses, public institutions, and the like. It is just in these cases that one learns how, amidst seemingly good hygienic circumstances, the conditions for an outbreak of typhoid fever are furnished by badly laid, insufficiently emptied, choked, or ill-ventilated drains, by leaking or over-filled cesspools, and the like; we see cases of typhoid, singly or in groups, beginning to occur, from the moment the noxious influences associated with these nuisances make themselves felt, and precisely

among those who occupy the rooms or buildings exposed to those influences in one way or another (of which more anon), and we find that the attacks cease to occur when the defects are repaired.[1] But evidence on a large scale for the importance of this etiological factor in the production of typhoid is furnished by the lowering of the typhoid sick-rate and death-rate in towns, through attention to the cleansing of fæcal matters from the streets, the houses, and the soil, and through an efficiently carried out system of drainage and sewerage; and this evidence comes mostly from the country which takes incontestably the first place among the States of Europe for its sanitary administration as laid down by statute—I mean England.

I take the following data from the report drawn up by Buchanan in 1866,[2] on the results attained up to that time in the improvement of the health of the inhabitants in several of the large towns of England, consequent on regulations for the public health. In nine towns the mortality from this disease had fallen 52 to 75 per cent. below the former level, in ten others 33 to 48 per cent., in Rugby 10 per cent., and in Carlisle 2 per cent.; on the other hand, it had risen in three towns—in Chelmsford 5 per cent., in Penzance 6 per cent., and in Worthing 23 per cent. Accompanying these statistical data is a statement by Buchanan, that drainage of the soil, provision for good drinking water, and other provisions of a like kind, had undoubtedly contributed to bring about the favorable results in the first group of nineteen towns; but that it was, above all, prevention of fouling the air (and the soil) by organic refuse that had uniformly the effect of producing a decline in the amount of typhoid; and that the exception of the second group of places was completely explained by their defective system of sewerage, which had permitted the sewer gas to penetrate into the houses. "It appears," he says at the end of his report, "that the four towns, where fever has not been greatly reduced, are so far from constituting an ex-

[1] Murchison gives a series of observations of this kind ('Continued Fevers'). Specially noteworthy are the facts relating to a school at Clapham in 1829, to Westminster School in 1848, and to the boys' school belonging to the Colchester Union. Among more recent observations are: those by Gillespie ('Edin. Med. Journ.,' 1870, May, p. 965) for Donaldson's Hospital, Edinburgh, in 1869; by Schmiedt ('Deutsche militär-ärztl. Zeitschr.,' 1875, p. 78) for a military barrack at Blankenburg i. H. in 1873; by Schwartz ('Arch. méd. belge,' 1875, Avril, p. 233) on an epidemic of typhoid in 1874 at the barracks in Liege; by Page ('Brit. Army Reports,' 1874, xv, p. 301) on an outbreak of the disease in 1874 at the Kildare barracks; and by Haviland ('Lancet,' 1876, Jan., p. 176) on the outbreak and epidemic prevalence of typhoid in 1875 in the well-appointed buildings of Uppingham School.

[2] 'Ninth Report of the Med. Officer of the Privy Council,' 1866, p. 43.

ception to the rule—that removal of organic impurity from the air has been followed by reduction of typhoid—that they even add strongly to the presumption that the rule is absolute and universal."

The sick-rate from typhoid in the several quarters of Hamburg before and after the introduction of sewers, forms another interesting contribution to the question before us. While, in 1876, the town proper was almost completely provided with a drainage-system, and the suburbs of St. Georg and St. Pauli partially so, the old cess-pit system still remained in the other (country) districts. According to the report of the inspector of public health,[1] the ratios of typhoid from 1872 to 1876, in 1000 of the population, were as in the following table:[2]

Table of the Typhoid Sick-rate per 1000 *Inhabitants in Hamburg and Vicinity.*

	1872.	1873.	1874.	1875.	1876.	Total.	Yearly Average.
Town of Hamburg .	3·65	2·29	2·54	2·41	1·40	12·29	2·46
Suburbs of St. Georg and St. Pauli .	2·91	2·52	3·40	3·05	1·86	13·74	2·75
Country Districts .	4·19	3·29	3·71	2·70	2·37	16·26	3·25

Virchow, who has collected information of the death-rate in Hamburg for the period from 1838 to 1869, points out[3] that the average mortality from typhoid in 1000 deaths from all causes, was:

For 7 years	(1838-44) before the town was sewered . .	48·5
9 „	(1845-53) while the sewers were being made .	39·5
8 „	(1854-61) after part completion of the sewerage	29·9
8 „	(1852-69) subsequently	22·0

From which it appears that the amount of sickness (estimated by the mortality) had fallen to less than half in thirty-two years.

Not less remarkable are the results arrived at Liévin[4] with respect to the mortality from typhoid at Danzig before and after the laying down of sewers. Whereas, in the period from 1863 to 1871, 360 persons in all died of the disease, or 70 a year, the mortality from 1872 to 1879 (or subsequent to sewering) was 221, or 27·6 a year, the co-efficient of mortality falling from 10·01 to 3·62. "The most remarkable

[1] 'Bericht über die med. Statistik des Hamburger Staates für das Jahr 1876,' p. 23.

[2] The figures do not agree altogether with the data communicated the year before by Medicinal-Inspector Herr Kraus to Herr Virchow, and published by the latter in his 'Gesammelte Abhandlungen,' ii, p. 437.

[3] L. c., ii, pp. 438, 470.

[4] 'Ueber die Sterblichkeit in Danzig in den Jahren 1863-79,' Danzig, 1879, p. 29.

diminution, amounting to 78 per cent., took place in the districts where deaths from typhoid had been most common, and which held the lowest place in almost all sanitary respects."

There can be scarcely any well-grounded doubt, then, as to the causal connexion between the origin and diffusion of typhoid, and influences exerted by putrefying excreta; but, at the same time, the doctrine developed by Murchison, and accepted by many later observers,[1] that the putrid decomposition-products of fæcal matter represent in themselves the typhoid poison, or that the disease is referable to a process originated by putrefactive matters (a "pythogenic" process, according to Murchison), appears to be altogether untenable. Apart from the consideration that the organic nature of the morbid poison is highly probable, if it be not definitely proved, there are unambiguous facts to show that the disease has very frequently occurred under circumstances where the influence of putrefying fæcal matters could not come even remotely into the question. If the specific poison were developed from this cause—one might unhesitatingly call it a commonplace cause—typhoid would be infinitely more frequent than, as a matter of fact, it is; and it is with justice that Folsom points out how thousands of people inhale the decomposition-products set free from fæcal matters, and drink the water polluted with excrementitial substances, without falling ill of typhoid. How often do we find not only single houses, but whole groups of houses in which the air of the living-rooms and sleeping-rooms is so charged with decomposition-products of putrefying sewage, that even a not very nice sense of smell is most unpleasantly affected by it; and yet there is no typhoid in these dwellings! The filthiest localities or quarters, in which there is not the remotest idea of a rational disposal of animal excrement, remain quite free from typhoid for years, and not unfrequently for dozens of years. However highly, then, we may rate the significance of the etiological factor here spoken of for the production of the disease, we shall always see in it only one link—unquestionably an important link—in the chain of causes upon whose influence, direct or indirect, the

[1] Tweedie, 'Lancet,' 1860; Prior, ib., 1870, Aug., p. 289, Sept., p. 327; Low, 'Brit. Med. Journ.,' 1880, Nov., 733; Don, ib., Nov., 737.

potency of some specific substance, the peculiar morbid poison, is more or less dependent.

§ 164. A Communicable Disease; Question of Autochthonous Origin.

The discussion of these matters brings me to a field of lively medical controversy, to the question of the so-called *autochthonous origin of typhoid*. For the settlement of this question, it is necessary in the first place to come to an understanding whether the disease is to be counted among the *communicable or contagious processes*. The very first observers, who made typhoid a subject of exact inquiry, were divided in their opinions as to the communicability of the disease. While Andral,[1] Chomel, and other French hospital physicians remained unconvinced of its communicability, on the ground of their hospital experiences, others, such as Leuret,[2] Bretonneau, Tardieu,[3] Fouré,[4] and above all Gendron, advocated the contagiousness of typhoid. Gendron not only expressed himself[5] in favour of the possibility of a conveyance of the disease, but he declared that the production of epidemics or endemics of typhoid, as well as the occurrence of sporadic cases, never took place except through direct or indirect communication. In this he was the precursor of Budd,[6] Gietl,[7] and others, who asserted that "the contagious nature of typhoid is the central point of interest in the history of the disease." Although this divergence of opinion is not yet reconciled (some hyper-sceptical persons still denying the communicability of the typhoid poison) yet the great majority of unprejudiced inquirers have arrived at the affirmative conclusion.

Not to mention the perverse stating of the question and defective logic in drawing inferences from observed facts, an important source

[1] 'Cours de pathologie interne,' 3 éd., Bruxell., 1839, p. 13.

[2] 'Arch. gén. de méd.,' 1828, xviii, p. 161.

[3] 'Journ. des connaiss. méd.,' 1835, Août.

[4] 'Journ. de la Soc. de méd. du Depart. de la Loire infér.,' 1837, p. 56.

[5] 'Journ. des connaiss. méd.-chirurg.,' 1834, Mars, Avril, Juli, Octobre.

[6] 'Lancet,' 1856, 1859, 1860, and 'Typhoid Fever, its Nature, Mode of Spreading, and Prevention,' Lond., 1873.

[7] 'Die Ursachen des enterischen Typhus in München,' Leipz., 1865.

of erroneous observation arose out of the circumstance that the field of inquiry was for the most part the far too extensive areas of great towns; whereas the question cannot be decided with certainty except within small circles of diffusion, particularly on the flat country and more particularly where the population is thinly scattered. It was precisely under the latter conditions that Gendron instituted his valuable inquiries; he illustrated the fact of communicability, or, as he says, contagiousness, particularly in instances where he was concerned with the appearance of the disease after the arrival of a patient suffering from typhoid at a locality lying quite isolated and hitherto quite healthy. The first cases of the sickness occurred in the immediate neighbourhood of the stranger, and its further diffusion in the place, or, it might be, beyond it, could be traced step by step; so that he got, in such a field of medical inquiry, as high a degree of proof of the etiological connexion between the first case and those that followed as could be anywhere attained. Many later investigators have arrived at the same conclusions as Gendron: including, in quite recent years, Cold,[1] according to experiences in Denmark; Homann and Hartwig, Collet, Eger,[2] and others from observations in Norway; the chronicler of the epidemic of 1875-76 in the Rhöngebirg;[3] Martin,[4] for the epidemic of 1857 at Titmoning (district of Laufen); Kerschensteiner[5] at Mering (Bavaria) in 1861; Lotholz[6] from several years' observations in the neighbourhood of Jena; Francis in India, Lee[7] from experiences in 1859 in the thinly populated county of Westchester, N.Y., Rothrock[8] from McVeytown, Pa.,[9] Pearce from Mechanicsburg, Ohio, Reeves (l. c.) from Virginia, and others.

As supporting the doctrine of the non-communicability of typhoid, particular stress used to be laid on the fact that cases of typhoid had occurred extremely seldom, or not at all, among hospital nurses and physicians, as well as among other patients treated in the hospital and even in the fever wards—cases which would necessarily have been referred to a conveyance of the disease from patients admitted with it. But more recent observations[10] have afforded evidence that conveyance

[1] L. c., p. 22. [2] Ll. cc.

[3] 'Correspondenzbl. des ärztl. Vereins in Thüringen,' 1876, p. 119.

[4] 'Bayr. ärztl. Intelligenzbl.,' 1858, p. 216. [5] Ib., 1861, p. 413.

[6] 'Beiträge zur Aetiologie des Ileotyphus,' Jena, 1866.

[7] 'Amer. Journ. of Med. Sc.,' 1859, Oct., p. 335.

[8] Ib., 1866, Oct., p. 574.

[9] 'Cincinnati Lancet,' 1865, May.

[10] Collected by Griesinger ('Infectionskrankh.,' 2nd ed., p. 147) from the Zürich Hospital; by Becker ('De typho abdom. epid.,' &c., Diss., Rostock, 1847) from the hospital of Rostock; by Jessen ('Der Abdominaltyphus im Altonaer Krankenhause,' &c., Kiel, 1869) from the hospital of Altona, and by Trier (l. c., p. 116) from the Copenhagen Hospital.

under those circumstance is by no means so rare as had been supposed; besides which, there are experiences made on board English ships of war of the importation of typhoid from the shore to the ships, and from these again to other ships.[1] So that, even although the seemingly positive results of the experiments to infect animals (rabbits) with the contents of the intestine or with the blood of typhoid patients[2] should not be substantiated, there cannot be a single doubt raised as to the communicability of typhoid. And the proof of the communicability of typhoid furnishes an answer decidedly in the negative to the question of the autochthonous origin of the typhoid virus, for everyone who holds, as a principle hitherto unshaken, the specificity of the acute infective diseases, and the corresponding specificity of the morbid poison proper to each. In whatever way we may regard the virus and its relations to the human organism, whether as a "miasm" or otherwise, it is capable of reproduction, and it must at all events be regarded as an organic body.[3] There are, no doubt, many observations on the occurrence of isolated cases of typhoid, or on the outbreak of epidemics, in which no conveyance or impor-

[1] Friedel, 'Die Krankheiten in der Marine,' Berlin, 1866, p. 201.

[2] Davaine ('Bull. de l'acad. de méd.,' 1873, No. 4), who inoculated rabbits with the blood of typhoid patients observed, that the animals died with *septic* manifestations; the infective experiments of Birch-Hirschfeld ('Allg. Zeitschr. für Epidemiol.,' 1874, i, p. 31) gave a very ambiguous result; those of Klein ('Report of the Medical Officer of the Privy Council,' 1876, new ser., vi, p. 80), of Bahrdt ('Archiv der Heilkde.,' 1876, p. 156), and of Moschutkowsky ('Centralbl. für die med. Wissensch.,' 1876, No. 11, on men and animals) were unsuccesful; while Letzerich ('Archiv für experimentelle Pathologie,' 1878, ix, p. 312) and Klebs (ib., 1880, xii, p. 231, 1881, xiii, p. 381) find proof positive of the conveyance of typhoid in their experiments with rabbits, and Tizzoni ('Annali univ.,' 1880, Feb., p. 97) in his experiments with dogs.

[3] The earlier inquiries on the "typhoid parasite" (micrococcus, bacterium, &c.), include those of Hallier ('Virchow's Archiv,' 1868, vol. 43, p. 287); Birch-Hirschfeld (l. c.); Eichhorst ('Arch. f. klin. med.,' 1874, xiv, p. 233); Klein (l. c., and 'Centralbl. für die med. Wissensch.,' 1874, Nos. 44-45); Feltz ('Compt. rend.,' 1877, lxxxv, p. 1288); Letzerich ('Virchow's Archiv,' 1876, vol. 68, p. 532, and l. c.); Fischel ('Prager med. Wochenschr.,' 1878, Nos. 4-5); Eberth ('Virchow's Archiv,' 1880, vol. 81, p. 58); and Tizzoni (l. c.). These have been followed by the recent work of Klebs (ll. cc.), who has found, uniformly present in considerable quantities in the intestinal infiltrations of typhoid patients as well as in the secondarily affected mesenteric glands, larynx, lungs, pia mater and kidneys, a minute fungus which he names "Bacillus typhosus."

tation of the disease from without could be ascertained. Observations of that kind must always be received with scepticism, inasmuch as it is often notoriously difficult to trace and to unmask the ways of conveyance of a disease, especially where intercourse is of the more active kind. Still, there are many observations to which this is not applicable, especially those relating to the occurrence of the disease in districts with very limited means of communication, or with a scanty and widely scattered population, where the facts are easily controlled and error as much as possible excluded. Of this class are the data of Metcalfe for Norfolk Island, Maclean for Ascension, Lange for Greenland, Hjaltelin and others for Iceland, and Ripley for the Fiji Islands. But, even in these and similar cases, I find no sufficient reason for assuming a spontaneous origin of the morbid poison. I am rather inclined to think that we are here concerned with the quickening of germs of disease which had remained long latent; and this conjecture appears to be all the more justified, that other infective diseases, such as cholera, yellow fever, plague, and the like, are known to exhibit the same behaviour. There is this difference, however, that, in contrast to the ubiquitous diffusion of typhoid, these are indigenous at only a few points of the globe, the poisons proper to them being able to survive outside their habitat for a relatively short time, and speedily perishing; so that, unlike typhoid, the disease when it reappears presupposes a fresh importation of the morbid poison.

§ 165. Mode of Diffusion of the Morbid Poison.

The reproduction of the virus of typhoid takes place, probably, in the intestine of the sick person. The poison, so produced, is discharged with the dejecta, and gets carried from place to place, partly by patients themselves, partly by their body-linen and bed-linen soiled by the evacuations, perhaps even by healthy individuals to whose clothes or to whose persons the virus may cling.

The fact that the disease is comparatively seldom communicated, even when a large number of typhoid patients

are crowded into one space, or that typhoid, as it is sometimes expressed, is contagious only in a feeble degree, together with the fact of its being associated in its origin and diffusion with the above-mentioned conditions of locality, justifies the assumption that it differs essentially, as regards communicability, from the properly contagious diseases, which are almost independent of external influences of that kind—such diseases as typhus, scarlatina, and smallpox. These facts seem to show that the virus eliminated from the intestine of the sick person is not yet capable of producing an effect of its own; that its potency, which may be represented as a kind of ripening, depends on certain external conditions; and that, among those conditions, the chief is the presence of putrefying organic excreta, and more especially, as it seems, the concurrence of the virus with putrefying excrement in the soil, in cesspits, or in badly flushed sewers and drains from privies and water-closets.

Cases of importation of the disease by means of linen or clothes are given by Gendron, Murchison (on an anonymous authority),[1] Huppert,[2] Schwab,[3] Proels,[4] and others. In all these cases, it is a matter of the disease breaking out in small and easily surveyed localities which had hitherto enjoyed perfectly good health, after the introduction of effects that had been used by typhoid patients during their illness, and particularly of things that had been soiled by the dejecta. The first cases of sickness occurred among those who had to do with the articles in the first instance. Attention has been called, in several quarters, to the comparative frequency of attacks in laundresses who had been washing linen soiled with the dejecta of typhoid patients.

Of the part played by *the soil in the development or diffusion of the typhoid poison*, we can only make conjectures for the present. In all probability, the soil serves as a breeding-place for the virus, which reaches it either from cesspools or ashpits or directly, and comes to maturity in it under the influence of the decomposition-processes undergone by the faecal matters which had percolated along with the virus or had accumulated previously. It cannot be denied, *a priori*, that fluctuations of the sub-soil water, or, in other words, the entrance of air into ground previously saturated and then

[1] L. c., p. 433. [2] 'Arch. der Heilkd.,' 1877, p. 197.
[3] 'Bayr. ärztl. Intelligenzbl.,' 1878, p. 40. [4] Ib., 1880, p. 145.

left dry, which follows therefrom, is particularly calculated to further putrefactive processes; and in this way may be explained the influence of fluctuations in the sub-soil water on the number of cases, or, in other words, the fact that typhoid reaches its highest point after a great fall of the sub-soil water, and its lowest after a considerable rise. The virus, having become potent, can now be carried from the soil into the atmosphere with the ascending current of air, of which risk, as Vogt surmises,[1] the height of the barometer will be an index; and it will so get introduced into the human organism with the breath. But the influence of fluctuations in the sub-soil water may be also expressed by saying that they exert a purely mechanical effect on the introduction into wells of the virus which had penetrated into the soil.

Liebermeister[2] formulates Buhl's law as follows: "When the level of the water in the wells rises, the amount of typhoid diminishes; when the water in the wells falls, typhoid increases." As he assumes, further, that the morbid poison comes to maturity in the soil and gets carried thence into the wells, and that the water drawn from wells is the proper vehicle of the typhoid poison, he would explain the coincidence of highest and lowest points of sickness with low and high levels respectively of the sub-soil water, by the fact that all the constituents dissolved or suspended in water will occur in larger quantity in spring water when the wells are low than when they are high. Buchanan[3] understands matters somewhat differently. Admitting that water stands at the same level in wells as in the soil round about, he further points out that all the water which gathers in the ground within a certain radius of the well, and often many feet above its level, from rain, from slops,

[1] 'Correspondenzbl. für Schweizer Aerzte,' 1874, iv, pp. 69, 212. Vogt ventures to conclude, from observations made in the epidemic of 1873 at Bern, that there is an increase in the amount of sickness with a falling pressure, and a decrease when the barometer rises, inasmuch as the gases in the soil issue forth the more copiously the lower the barometric pressure. Pribram and Popper (l. c., p. 16) have found, on comparing the barometric fluctuations with the amount of typhoid sickness: (1) that there is no apparent connexion between the maximum levels of the disease and of the barometer; (2) that a rapid increase in typhoid has often followed when the barometer has been at its lowest readings, steadily decreasing again with a continuance of moderate pressure; and (3) that an increase in the number of cases has corresponded to great fluctuations of the mercury, while there has been a decrease when the fluctuations were slight.

[2] 'Deutsche Klinik,' 1866, p. 90.

[3] 'Med. Times and Gaz.,' 1870, March, p. 283.

from cesspools, &c., will run off into the well, and that these percolating supplies of water will, accordingly, reach the well in all the larger quantity the lower the water-level in the well—that the various matters will be carried down most readily when the sub-soil water is on the fall, and the contents of the well be contaminated most under these circumstances. It is obvious that the explanations of Liebermeister and Buchanan as to the significance of fluctuations in the sub-soil water for the occurrence of typhoid, apply only to those cases where drinking-water forms the vehicle of the morbid poison.

Notwithstanding the obscurity which still hangs over all these questions—an obscurity increased by the fact that Buhl's law, as it seems, is applicable to certain places only, having no relevancy for others—no one can deny the importance of the soil as the breeding-place of the typhoid poison. No doubt typhoid develops under circumstances where any influence of the soil is not only highly improbable, but even excluded as an etiological factor altogether, as in epidemics in rooms. But those cases are by no means in contradiction of the theory ; they serve rather to corroborate it, inasmuch as the same conditions that cause or assist the typhoid poison to ripen or acquire potency in the soil, may be met with also outside the soil.

"What the soil is on the large scale," says Lindwurm[1] very justly, "the same on a small scale are also the floors of rooms, the walls of houses, the drains of privies, and the like. Just as it matures at some depth in the ground, so also may the typhoid germ obtain the necessary conditions for its growth in a seam or cleft in the flooring of a room, or in the loosened mortar and sand between stones and slabs."

§ 166. Acclimatisation ; no Racial Exemptions.

Individual predisposition to the disease is a decidedly important point in the production of typhoid. Any discussion of the influence exerted in this way by age, sex, or mode of living, I consider to lie remote from my task ; and I have to mention only one fact relating hereto, an especially interesting one, and one that holds good everywhere, but more especially in tropical and subtropical

[1] 'Bayr. ärztl. Intelligenzbl.,' 1873, p. 232.

latitudes—I mean the protection which is afforded against typhoid by *acclimatisation*.

It had been pointed out by Petit and Serres[1] that many of the patients treated by them for typhoid were strangers who had resided only a short time ("nouvellement arrivés") in Paris. This fact was afterwards confirmed by Andral[2] and Louis,[3] the latter showing that in 129 cases there were 120 persons who had not resided more than thirty months in the capital, and that among 44 patients who succumbed to the disease, there were only two whose residence there amounted to over two years and a half. This predisposition to typhoid of non-acclimatised persons came out more markedly in the French garrison towns among fresh drafts of troops. Mondret[4] had pointed this out as early as 1823, on the ground of experiences at Le Mans; a selection of similar observations in subsequent years (especially 1873 and 1874), has been published by Colin;[5] and the same experiences have occurred in the garrison of Brussels in 1871 and 1873-74.[6] Trusen[7] also mentions the fact that in the epidemic of 1833 in Posen, it was chiefly the newly-arrived recruits that suffered. In London, among 1978 typhoid patients received into the Fever Hospital during fourteen years, there was 631, or nearly one-third, who had not completed a year's residence in the city;[8] in Dundee the same circumstance was even more marked.[9] In the General Hospital at Munich during the winter of 1856, and the academical year 1858-59, there were in all 365 admissions for typhoid; of these 187 (or just one-half) had lived in Munich not quite six months, and 66 not quite a year; and among the whole number there were only 6 who had been born in Munich.[10] For Malta, we are told by Marston (l. c.) that by far the larger number of typhoid cases were met with among the troops newly arrived from England.

This circumstance comes out still more strikingly in tropical and subtropical regions, where the disease is almost limited to strangers, and mostly to new arrivals. This holds good, first of all, for India, according to the unanimous judgment of observers;[11] in the 'Sanitary Reports' for the Presidency of Bengal,[12] 1872, we read: "That enteric

[1] L. c., p. 127.
[2] 'Cours de pathol. interne,' 3rd ed., Brux., 1839, p. 13.
[3] L. c., 1841, i, p. 357.
[4] 'Journ. gén. de méd.,' 1824, vol. 87, p. 318.
[5] L. c., p. 65.
[6] Molitor, 'Arch. méd. belge,' 1872, Mars; Gys, ib., 1874, Avril, p. 223.
[7] 'Casper's Wochenschr. für die Heilkde,' 1835, p. 337.
[8] Murchison, p. 421.
[9] Maclagan, 'Edin. Med. Journ.,' 1867, Oct., p. 314.
[10] Vogel, 'Klin. Untersuch. über den Typhus,' Erlang, 1856, p. 6; Zahler, 'Bayr. ärztl. Intellbl.,' 1861, viii, p. 311.
[11] Bryden, Moffat, Hanbury, Don, and others.
[12] 'Brit. Army Report' for the year 1872, xiv, p. 147.

fever in India, as in other parts of the world, is a disease from which strangers visiting an infected locality are much more liable to suffer than the permanent inhabitants of that locality, may be considered beyond doubt." Of 162 cases of typhoid in the Madras army which were notified in the years 1872-73, there were 99 affecting persons who had not served two whole years in India; in the Bengal army, the proportions of sickness from typhoid were 9·7 and 1·9 of the total strength among the troops who had resided in the country one to two years and over two years respectively (Ker Innes, l. c.). Similar observations as to the especial prevalence of the disease among newly-arrived or non-acclimatised persons, come to us from Tahiti,[1] New Caledonia,[2] Mauritius, the West Coast of Africa (*Chassaniol*), Senegambia (*Defaut*, p. 124), Algiers,[3] the Antilles,[4] Monterey (California),[5] and other places. In the 'Sanitary Reports,' of the English Royal Navy also, attention is drawn, as we have already seen, to the frequent occurrence of typhoid among the crews of men-of-war in tropical harbours.

This relative immunity of acclimatised persons from typhoid is not dependent on *peculiarities of race or nationality*, whether in temperate latitudes or in the tropics. Negroes, who are well known to suffer in their native countries much more rarely from typhoid than non-acclimatised persons living there, become subject to the disease in America in no less a degree, *cæteris paribus*, than the white race.

Attention had been called by Tidyman[6] to the frequent occurrence of "typhus fever" (certainly typhoid and not typhus) among the negroes of the Southern States of the Union, who were almost exempt from malarial diseases; of 43 cases of typhoid treated by Sutton in Georgetown, Ky., in 1846, 13 were in negroes;[7] Lewis states that hundreds of negroes died of typhoid in Central Alabama in the winters of 1835-36 and -37; and Gaston[8] (l. c.) speaks of the very frequent occurrence of the disease among the negroes of South Carolina.

Just as little can the predisposition to typhoid in newly arrived persons be explained by the change in their mode of

[1] Notice in the 'Arch. de méd. nav.,' 1865, Oct., p. 282.

[2] Brun, de Rochas, Charlopin, p. 21.

[3] Haspel, 'Maladies de l'Algérie,' Paris, 1850, ii, p. 428; Masse, Frison, and others.

[4] Dutroulau, p. 35; Brassac, 'Arch. de méd. nav.,' 1865, Mars, p. 227; Carpentin, ib., 1868, Sept., p. 220, and 'Étude,' Par., 1873, p. 41; Batby-Berguin, l. c., p. 42; Rufz.

[5] King, 'Amer. Journ. of Med. Sc.,' 1853, April, p. 389.

[6] 'Philad. Journ. of Med. and Phys. Sc.,' 1826, xii, p. 319.

[7] Bartlett, l. c., p. 127.

[8] 'New Orleans Med. Journ.,' 1844, i, p. 417.

life, by excessive bodily fatigue, or any other external cause. In my opinion, we are met here by the same phenomenon which may be observed in the case of yellow fever, although in a much more marked degree in the latter, tending to show that the protection acquired through acclimatisation is by no means dependent exclusively on having gone through the disease once before. The immunity from typhoid acquired through acclimatisation is unquestionably much more perfect in tropical and subtropical regions than in higher latitudes. Whether, and how far, a tropical climate has an influence of its own in this respect; whether, as Bryden conjectures, the unaccustomed temperature of the tropics predisposes the new arrivals in India to the disease; or whether, conversely, a continuous residence in the tropics lessens the predisposition to the sickness—these are questions which must remain unanswered for the present.

§ 167. Drinking-Water and Milk as Vehicles of the Poison.

The media which act as *carriers of the morbid poison* —which serve to introduce it into the human organism— can be no other, obviously, than the air in which the virus is suspended or the articles of food to which it clings. There can be no doubt that the infection takes place sometimes in the one way, sometimes in the other. It is highly improbable, considering how very closely the foci of disease are circumscribed, that the infecting substance can be diffused by the wind to any great distance. At the same time, the notion that the virus, disengaged in or upon the ground (or some one of its equivalents, as in p 674), gets carried off by the ascending or horizontal current of air and so conveyed into the human organism with the breath, is not only postulated in order to explain the origin of the disease in all those cases where another mode of introduction cannot be proved; but it is even based upon a good many observations, in so far as events of that nature can be proved at all. Especially noteworthy are the cases where it can be proved that sewer gases (with which the morbid poison must have been mixed) have found entrance into living.

rooms, and just those persons have sickened, who had been directly and principally exposed to these emanations, while other residents of the locality, who had not come under the noxious influence, had remained exempt from the disease.[1]

But a more frequent and more extensive source of infection is water for culinary purposes, which has been contaminated by the typhoid poison. I take it that few points in the etiology of typhoid are so certainly proved as the *conveyance of the morbid poison by drinking-water*, or by food contaminated with infected water. Although all the observations made on this point, and published to the profession, do not carry conviction equally, yet, in many of them, the causal connexion between the working of that noxious influence and the development of the disease comes out so clearly, that there can be no well-grounded doubt on the matter; if facts of that kind do not warrant conclusions as to the etiology of disease, then there is an end of all etiological proof. If, in a given locality, a number of cases of illness suddenly occur, exclusively confined at the beginning of the epidemic to one portion of the population, to those, namely, who had got their water-supply from one spring, one conduit, or one pump; if it be proved, further, that this water-supply has been contaminated by sewage, or by the typhoid poison contained in the sewage; if it be shown, still further, that the affected inhabitants have everything else in common with the exempted (the same kind of weather, the same dwellings, the same soil), differing from them in the conditions of their existence only in the use of the impure drinking-water; if, lastly, the epidemic come to an end, as it has been found to do in many cases, with the closing of the poisoned spring;—then, in my opinion, the inference, *post hoc ergo propter hoc*, is warranted concerning the origin of the disease so absolutely that even the most rigorous scepticism must be satis-

[1] Instructive examples of this kind are given by Murchison (l. c., pp. 437-45), from several English institutions, by Liebermeister ('Arch. für klin. Med.,' 1870, vii, pp. 180-81) from the Basel hospital and a few villages in the neighbourhood of Basel, by Haviland ('Brit. Med. Journ.,' 1876, Jan., p. 138) from Uppingham School, by Maclagan (l. c., p. 310) from the Dundee neighbourhood, and by others. An epitome of all the observations of that kind published since 1866 will be found in my summaries on typhoid in the 'Jahresberichten über Medicin.'

fied with it. I shall confine myself to giving a few of the most recent observations, which furnish incontestable proofs, as it seems to me, of the causal connexion between the use of drinking-water specifically tainted and the production of the disease.

In the often-quoted epidemic of typhoid at Stuttgart in 1872, which was proved to be a case of fouling of the town's water-supply by sewage, the diffusion of the disease extended just as far as this water-supply reached; and that fact could be established with every certainty, as we learn from the report of Burkart,[1] even within single houses. Thus, in the house No. 1, Feuerseeplatz, the residents on the first and third floors, who got their water by contract from private wells in the neighbourhood, were completely exempt from the disease, whereas on the second and fourth floors, where water from the town mains was supplied, several cases of typhoid occurred.

De Renzy[2] shows, in a statement of the health of Millbank Prison in London, which had been used for convicts since 1816, that although it had been at one time notorious for the endemic prevalence of typhoid, it had been almost entirely exempt from that disease after being supplied with pure water, although no further changes, structural or other, had been made in it. Up to 1854 the water-supply consisted of filtered Thames water; since that date the building has been supplied with water from the artesian well in Trafalgar Square; and, from the middle of that year, down to April, 1872, or a period of nearly nineteen years, there had been only three deaths from typhoid in the prison, one each in 1855, 1860, and 1865; and, since 1865, not a single case of typhoid had been observed in it.

In Bern, in the winter of 1873-74, there sickened of typhoid, according to the official report of Wyttenbach,[3] 355 persons out of a population of (in round numbers) 38,000. The cases occurred in good and bad dwelling-houses almost equally, and there were no special differences manifested in the amount of sickness in the various occupations or classes. No pathogenetic significance could be laid upon meteorological influences, or upon the state of the soil and of the sub-soil water, inasmuch as the parts of the town affected by the disease and those spared by it had all these factors in common. The one difference between them lay in the source of their water-supply. While hardly one-third of all the inhabitants of the town got their water from the main, fed by the so-called Gasel spring, it was shown that 79 per cent. of the sick were consumers of this water, and that almost all the cases of typhoid at that time in the outskirts of the town occurred in quarters where the

[1] 'Württemb. Correspondenzbl.,' 1872, p. 85.

[2] 'Lancet,' 1872, June, pp. 787, 820.

[3] Published in abstract in the 'Correspondenzbl. für Schweizer Aerzte,' 1877, p. 529.

Gasel water was used. An examination of the waterworks showed that two badly-constructed reservoirs had been polluted by sewage.

The following account of a severe outbreak of typhoid in a boarding-school at Mansfield, Pioga County, Pa., in the autumn of 1874, is given by Brown.[1] Of sixty-nine boarders twenty-eight took the disease more or less severely. The drinking-water of the institution was taken from an artesian well 140 feet deep, but there was also a pump directly behind the house, in close proximity to a cesspool and with a drain running past it. Although the water from the artesian well was excellent, many of the inmates of the institution preferred the water from the pump, which was in the highest degree contaminated. The servants, with one exception, took their water-supply solely from the artesian well, and they escaped the disease, all but one—the person who preferred the pump water. A workman, also, engaged at the time of the outbreak in working in the hall of the building, who had used the water but was otherwise unconnected with the institution (he lived across the river), fell ill with typhoid. After the nuisance was stopped no further cases occurred in the house.

In the village of Gunnislake a resident, who had returned from a visit to Liverpool, sickened with typhoid a few days after his arrival; and this case was followed in a very short time by 143 other cases, which all occurred in the houses in the centre of the village, while there was not a single case anywhere round about. The distribution of the disease, as Blaxall has shown,[2] corresponded exactly to the water-supply for the village from a particular reservoir, to which the intestinal evacuations of the first patient had probably got access. Among those inhabitants of the village who got their water-supply from other sources, only isolated cases occurred subsequently.

Towards the end of 1879 an epidemic of typhoid broke out at Caterham and Redhill (Surrey) so abruptly that, within the first fortnight, there were 47 cases in 35 houses of the former place, and 132 cases in 96 houses of the latter. The water-supply in both places was provided for partly by a service main situated in Caterham, and partly by private wells or rain-water cisterns. Of 558 houses in Caterham 419 got their supply from the main, as well as 924 of the 1700 houses in Redhill. The medical inquiry into the epidemic, conducted by Thorne,[3] went to show that both places had been free from typhoid for many years, that the disease had fixed upon well-appointed villas and upon the dwellings of the poor without distinction, that there could be no question of any common injurious influence due to cesspools, drains, and the like, inasmuch as in both places there were very various systems of filth-removal, the houses provided with water-closets being implicated in the epidemic not less than those with privies or earth-closets. On the other hand,

[1] 'Philad. Med. Times,' 1875, Nov., p. 80.

[2] 'Lancet,' 1876, Sept., p. 328.

[3] 'Ninth Annual Report of the Medical Officer of the Local Government Board,' 1879-80, Lond., 1880, p. 78.

it appeared that in the first series of 47 cases at Caterham, 45 occurred in houses supplied with water from the main, and that the remaining two patients had not only been frequenting houses so supplied, but could be proved even to have made free use of the conduit water. The inquiry at Redhill led to a similar result. Of the 96 houses first affected 91 had their water-supply exclusively from the main, and in the other five houses also, use had been made of that water from time to time. As to the befouling of the reservoir, Thorne gives the following explanation: The company which owns the Caterham Waterworks had early in January commenced excavations with a view to enlarging their reservoirs, in the course of which a perpendicular shaft of considerable depth was sunk down to the conduit. Among the labourers at the bottom of the shaft was a man who, as it afterwards transpired, had contracted typhoid at Croydon, where it was then epidemic, and during the first days of his illness he continued his work in the shaft. His motions being very frequent and profuse, it was impossible for him every time to leave the shaft, so that he eased himself at the bottom of it, and the dejecta passed direct into the conduit. *The outbreak of the disease took place simultaneously at Caterham and Redhill exactly fourteen days after the water-supply had been befouled in this manner.*

In the small town of Nabburg (Upper Palatinate), with 270 houses and 1900 inhabitants, where typhoid had been extremely rare for a long series of years, the wife of a labourer sickened of that disease, according to the account of Proels,[1] in the end of May, 1880, and eight days later her husband and her sister. The husband had shortly before brought home from Schmidgaden, his native place, where typhoid was then epidemic, among other articles a bed that had been slept on during her illness by a sister who had died of typhoid. About a fortnight after those three persons sickened, new cases of typhoid appeared in the row where their house was situated; these quickly multiplied, so that within the next two weeks thirty-five of the seventy-seven residents in that row had fallen ill. The inquiry into the local circumstances resulted in the following explanation: Opposite the house where the first three cases occurred, the pipes that supplied the street with water ran through a slop-puddle which received the soakage from the dungheap and the yard of the house. In one of the pipes which lay quite near the surface, hardly covered by earth and rubbish, an opening was found just at the place where the slops ran over; the hole was imperfectly stopped with rotten wood, and some part of the slops must have leaked through. Besides this pipe, there was another also running through the puddle, which was in so defective a state that the slops had free access to it; and from those two pipes the water thus befouled passed into a cistern close at hand, from which most of the residents in the street took their drinking water. It was shown, finally, that the dejecta of the first three patients had partly been thrown upon the dungheap in the yard of the house,

[1] 'Bayr. ärztl. Intelligenzbl.,' 1880, No. 14.

but had been chiefly emptied at the point where the slops ran off, so that after flowing over the street a few paces they entered the water-conduit direct. The notion that this befouling of the drinking-water had given rise to the outbreak of the disease is confirmed by the rapidity with which it spread, in this as in all other cases; and further by the fact that three out of the seventeen houses in the lane, which got their water from another source, as well as the inmates of the poors-house at the end of the lane, who were supplied with water from a pump in the yard of the institution, continued entirely exempt from the disease. There is a still further confirmation in the fact that the epidemic came to an end in the lane after the water-pipes were repaired.

Among articles of food infected by specifically-tainted water, which have served as carriers of the typhoid poison and have thereby occasioned the spread of the disease, *milk* has chiefly arrested the attention of observers hitherto; and more particularly the English medical officers of health have furnished interesting information on this subject.

Ballard[1] gives an account of an epidemic of typhoid in the summer of 1870, in a small district of Islington (London). In sixty-seven perfectly healthy houses, inhabited by families in good circumstances, 168 cases of typhoid occurred in rapid succession; whereas in the other parts of the suburb only twenty cases occurred. Almost all the cases occurred in families who got their milk from one dealer, who had suffered himself from typhoid in the first instance, and in whose family seven other persons had fallen victims to the epidemic. This man's business extended to about a mile from his house; and of the 2000 families resident within that area 142 got their milk from him, in seventy of which, at the lowest estimate, cases of sickness and death from typhoid occurred. Ballard showed that there was a well on the milk-dealer's premises, highly befouled by slops running into it; from this well water had been pumped to rinse the cans in which the milk was carried, and it had probably been used sometimes to water the milk.

A special interest was excited in an epidemic of typhoid which originated under similar circumstances in the summer of 1873, in one of the most fashionable quarters of London, including the parishes of St. Marylebone, St. John's Wood, and St. George's, wherein an unfortunate lot fell to the family of Murchison, to whom we owe so much of the doctrine of the typhous diseases, and who materially helped to elucidate the particular incident.[2] In this case also it was an affair of a large number of cases in numerous houses scattered over an extensive district, and inhabited almost exclusively by well-to-do families. The one thing

[1] 'Med. Times and Gaz.,' 1870, Nov., p. 611.

[2] Notices of the epidemic occur in the 'Brit. Med. Journ.,' 1873, Aug., Sept., and 'Lancet,' 1873, Aug., Oct.

that they had in common was that they got their milk from one Dairy Company. In about ninety families some 300 cases of typhoid were made known within four weeks (from the middle of July to the middle of August). The inquiry showed that a farmer who supplied the company with milk had taken typhoid and had died, and that the cess-pit into which the dejecta of the patient had been thrown communicated with the well of a pump whence water had been taken for cooking purposes and for cleansing the milk vessels, perhaps, also, for diluting the milk.

A farmer in the neighbourhood of Bergen sickened of typhoid on the 18th of August, 1872, and four days later his maidservant. During the illness of those two, the milk obtained on the farm was taken by the wife to customers in the town, to twenty-two families in all, and in eight of these the disease broke out almost simultaneously (between the 28th of August and 3rd September). That it was not the man's wife who acted herself as the carrier of the infective matter follows from the fact that, in one of the houses, all the children took typhoid, although the woman had never come into contact at all with the inmates.[1]

The following account is given by Lübe[2] of the spread of typhoid by infected milk in the small town of Plön in Holstein in the summer of 1875. The cases were scattered over the whole place in particular families, as well as in the families of the officials belonging to the College for Cadets situated on an eminence—the officials occupying the two wings of it—while among the 126 cadets themselves not a single case occurred. The state of the soil, of the sub-soil water, and of the drinking water, as the author shows, could not come into the question of the diffusion of the disease; that which was common to all the families who had a visitation of the sickness was the source of the milk-supply from a farm where cases of typhoid had occurred. (In only two cases no direct infection by this channel could be proved for certain, and in a third case the sickness seems to have been conveyed in some other way.) In the yard of the farm was a well, highly befouled by refuse, from which the water was taken for rinsing the milk-vessels. How the typhoid poison came into the well could not be proved with certainty. After the 5th of September the residents of the town ceased to purchase milk from that farm, all except one family; and there were no further cases except in a child belonging to the family in question, for whom milk had been procured from the farm as before.

At Worthing, in Sussex, according to the account of Kelly,[1] a young man took ill of typhoid on the 24th September, 1879, having arrived from London a few days before. The dejecta passed into a leaking drain, from which the sewage escaped into a well belonging to this man and his neighbour. The neighbour was a milk dealer, and he used the water from the well for household purposes. About a fortnight after the man fell ill, cases of typhoid appeared in various parts of the town, and there were

[1] Holmboe, 'Norsk. Mag. for Laegevidensk.,' 1873, p. 654.
[2] 'Allgem. Zeitschr. für Epidemiol.,' 1876, ii, p. 298.
[3] 'Brit. Med. Journ.,' 1880, Dec., p. 933.

more in the weeks following; these cases occurred solely in houses which got their milk from the dealer in question. On the 4th of November the contaminated well was closed, and on the 9th of November the last case occurred which could be referred to that source.

The extremely abrupt outbreak of the epidemic is noteworthy in all these instances of infection of drinking water; as well as the fact that, in those epidemics where the time of befouling of the well or conduit is made out, the consequent cases usually occurred after ten to fourteen days, or an interval corresponding to the period of incubation of the disease. It is also noteworthy, that in the cases induced by infected milk, it is principally women and children who have been attacked, or those who are the chief consumers of the article.

In view of these and many similar observations,[1] all objections which may be raised against the notion of typhoid being spread directly or indirectly by drinking-water must, in my opinion, fall to the ground; all the more so as they are of a purely negative kind and mostly based on the erroneous assumption that the supporters of that opinion would deny every other mode of conveyance of the disease. It must be conceded, as I have already said, that infection can be produced also by means of the air, as a carrier of the morbid poison, although the arguments brought forward in favour of that opinion are far from possessing the same degree of cogency as those in favour of conveyance by drinking-water. Again, the objection often raised, that, if the partaking of water befouled by sewage could produce typhoid fever, the disease would be infinitely more common than as a matter of fact it is—this objection has a radical error underlying it, inasmuch as it is not the sewage by itself that has a disease-producing effect, but the sewage by virtue of containing dejecta charged with the typhoid virus.

§ 168. Antagonism between Malaria and Typhoid.

I have still to touch, in a few words, on the connexion between *typhoid* and *malarial diseases*, the much debated question of their mutual exclusiveness, or the

[1] A tolerably complete collection of all the facts published about the diffusion of typhoid by infected water and milk since 1866 will be found in the *résumés* drawn up by me on the Acute Infective Diseases in the 'Jahresberichten über Medicin.'

antagonism which those diseases show one to another *in time and place*. Boudin[1] claims to have been the first to maintain, on the ground of observations made by himself and other French physicians in Algiers, France, and Italy, that typhoid rarely occurs in regions with endemic malaria, for the reason, as he believed, that a prolonged residence in a malarious region takes away the predisposition to typhoid in those thus acclimatised, or, in other words, in persons exposed to the continuous influence of malaria. This opinion afterwards found as many opponents as adherents; and the question of typhoid on the large scale being excluded from malarious regions is still undecided at the present day, while it is for the most part stated and argued in a sense different from that of Boudin.

The first support to Boudin's theory came from the side of several Belgian physicians, such as Woets[2] and Waldack,[3] who pointed out that, since the draining of the ground in the neighbourhood of Dixmude and Ecloo, the malarial fevers, which had been endemic there up to that time, had disappeared, while typhoid, which had been of rare occurrence before, had come in their place. Meynne[4] also stated subsequently that typhoid occurred much more rarely in the low-lying malarious regions of Belgium than in the hilly districts of the country; to the same effect are many statements from the Netherlands, such as that of Beduin[5] from Zeeland, one of the worst malarious districts, where, in the town of Middelburg only 275 cases of typhoid came under observation from 1860 to 1868, or 30 in the year. A kind of mutual exclusiveness of the two diseases in a locality has been manifested on the largest scale in the United States; here also it has happened that malarial diseases have become much less common in many parts of the country with progressive drainage and complete cultivation of the soil, while the amount of typhoid, hitherto a disease of rare occurrence, has risen in proportion. To this effect we have, first of all Flint's statements from Buffalo;[6] then Drake asserted that this relation between the two diseases could be seen at many points all through the Mississippi Valley;[7] and there are more recent confirmatory accounts by Reeves

[1] 'Essai de géogr. médicale,' Paris, 1843, p. 83.

[2] 'Annal. de la Soc. méd. de Brnges,' i, p. 17, and 'Journ. de méd. de Bruxell.,' 1844, Mars, p. 120.

[3] 'Annal. de la Soc. de méd. de Gand,' 1847, p. 69.

[4] 'Topogr. méd. de la Belgique,' Bruxell., 1865, p. 388.

[5] Nederl., 'Tijdschrift voor Geneesk.,' 1871, i, p. 357.

[6] 'Buffalo Med. Journ., 1847, Feb.

[7] L. c., ii, p. 447.

[8] L. c.

for Virginia, by Bland[1] particularly for the eastern parts of that State, and for New Jersey,[2] Pennsylvania,[3] and other localities.

On the other hand, it is alleged by Frison, Arnould and Kelsch, and others, that in Algiers, the original home of the Boudin theory, there is no such antagonism existing between malarial diseases and typhoid, at the present date at least, and that both diseases occur together in time and in place. Jacquez[4] observes that, in the arrondissement of Lure (Haut-Saône), a pronounced malarious region, typhoid has been several times epidemic; in like manner Fleury[5] says that typhoid is often met with in the malarious parts of the Vendée; and Janssens[6] denies the alleged antagonism for the "polders" in the district of Ostend. From the United States there are accounts by Grant[7] from Memphis (Tennessee) and by Reyburn[8] from Missouri, which also tell against the theory of Boudin. Leconte[9] has pointed out that malarial fever and typhoid are equally common in the countries of the Lower Danube; Smart[10] says that in Hong-Kong both diseases have been epidemic from time to time side by side; and Bourel-Roncière gives the same account of the relations of the two diseases in Rio de Janeiro. It is further noteworthy, that at the time of great pandemics of malaria, such as those of 1826-30 and 1846-48, typhoid was observed as an epidemic at many places along with the prevalent malady. It is, however, a particularly noteworthy circumstance that the endemic and epidemic occurrence of typhoid in malarious regions has lately given rise to the notion of typho-malarial fever, as a hybrid disease-form.

All these observations directed against the theory of Boudin merely serve, in my view, to prove that the latter is not universally applicable. The positive facts, especially the experiences on a large scale in Belgium and the Netherlands, and in the United States, lose none of their force. They may be interpreted, indeed in a sense not quite that of Boudin's. They furnish evidence, it seems to me, that the complex social revolution, in a population which has grown and become crowded in proportion as the sources

[1] 'Transact. of the West-Virginia State Med. Soc.,' 1871.

[2] 'Transact. of the New Jersey State Med. Soc.,' 1861.

[3] In almost every annual volume of 'Transact. of the Pennsylvania State Med. Soc.' from 1865 to 1877.

[4] 'Gaz. méd. de Paris,' 1845, p. 461.

[5] 'Des fièvr. intermitt. du marais de la Vendée,' Paris, 1874, p. 31.

[6] 'Annal. med.-chir. de Bruges,' ix, p. 17.

[7] 'Amer. Journ. of Med. Sc.,' 1853, July, p. 94.

[8] 'Transact. of the Amer. Med. Assoc.,' 1857, viii, p. 442.

[9] L. c.

[10] 'Transact. of the Epidemiol. Soc.,' i, p. 210.

of malaria have disappeared from the soil before drainage and cultivation, has its dark side as well as its bright; and that its effects are felt not least in things hygienic, and above all in an increased amount of the kind of sickness which is so closely bound up with changes in the habits of the people.

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